Presented by Mrs. Burridge

from the Library of the late

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and Toxicology.

November 1924.

FORENSIC MEDICINE

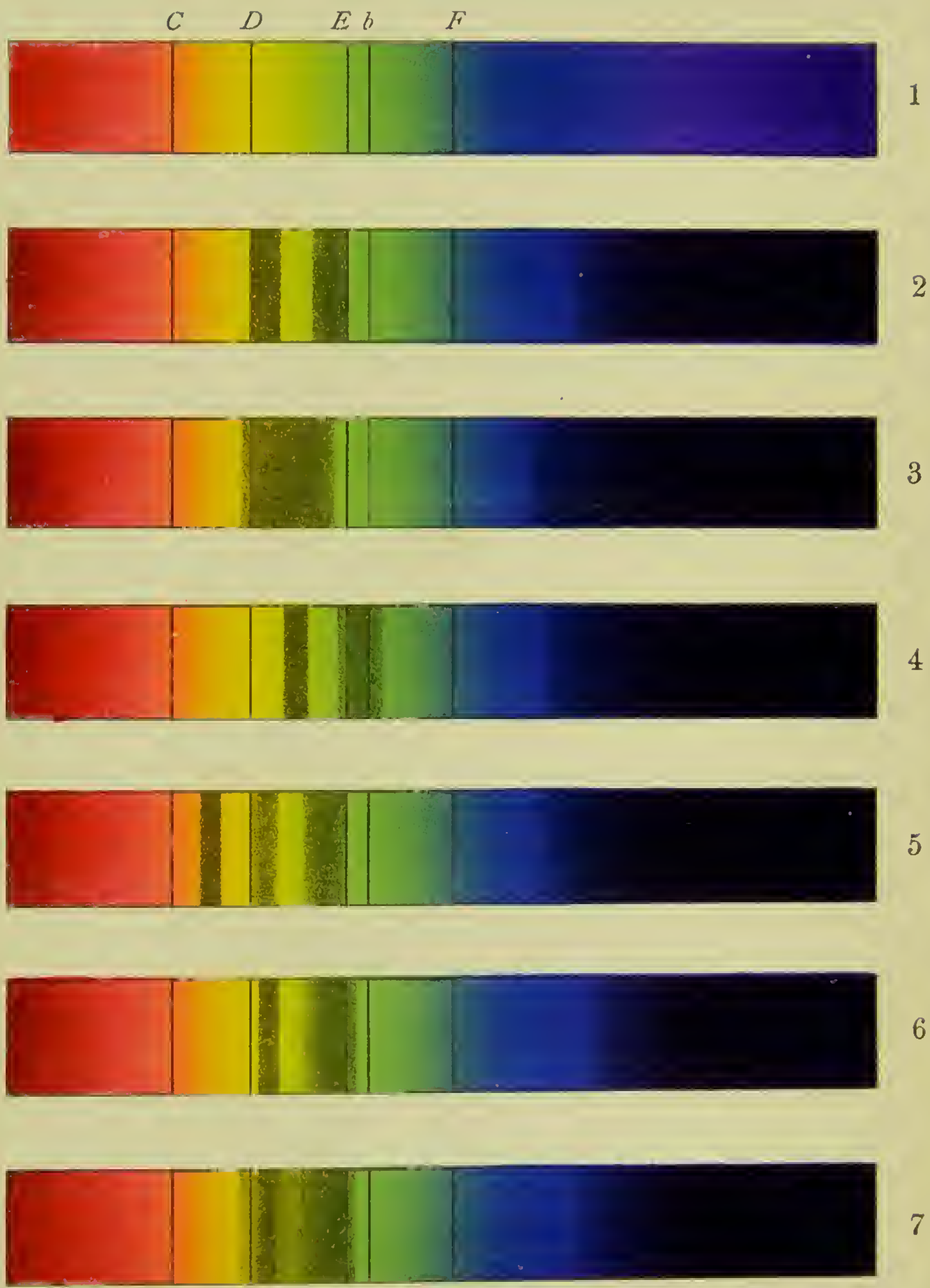
AND

TOXICOLOGY

VOL. I.

Profession -
"a trade which is organised, incompletely no doubt but genuinely, for the performance of function. + + + + +
It is a body of men who carry on their work in accordance with rules designed to enforce certain standards both for the better protection of its members & for the better service of the public.
+ + . + + + +
All professions have some rules which protect the interests of the community & others which are an imposition on it.
In its essence is that it assumes certain responsibilities for the competence of its members & the quality of its wares & that it deliberately prohibits certain kinds of conduct on the ground that though they may be profitable to the individual they are calculated to bring into disrepute the organisation to which he belongs. + + + +
The conception implied in the word "unprofessional conduct" is, therefore, the exact opposite of the theory & practice which assume that the service of the public is best secured by the unrestricted pursuit on the part of rival traders of their pecuniary self interest within such limit as the law allows."
(R.H. Tawney "The Sickness of an Acquisitive Society"
p 49 et seq.)

BLOOD SPECTRA.



1. Solar Spectrum.

2. Oxyhæmoglobin.

3. Reduced Hæmoglobin.

4. Reduced Hæmatin. or Hæmochromogen..

5. Methæmoglobin.

6. Co-Hæmoglobin.

7. Co-Hæmoglobin and Reduced Hæmoglobin.

Danielsson & Co., London, lith.

TEXT-BOOK

OF

FORENSIC MEDICINE

AND

TOXICOLOGY

BY

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IN TWO VOLUMES—VOL. I.

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PREFACE

This work has been written as a text-book for students of medicine, and as a work of reference for practitioners in connection with medico-legal cases. The author has availed himself of the material present in the works of Casper, Taylor and Stevenson, Reese, Blyth, Dixon Mann, and others, and has always endeavoured to make reference to such assistance in the text. No pains have been spared in the effort to bring all sections of the book up to date. Special care has been given to the section on Toxicology, and it is hoped that the directions for the treatment of cases of poisoning will prove useful to practitioners.

The author has to thank his friend Mr. Jackson Clarke, Pathologist to St. Mary's Hospital, for kind assistance and advice in connection with the chapter on the making of post-mortem examinations for medico-legal purposes, and his friend and colleague Dr. Gow, Physician Accoucheur in charge of Out-Patients in St. Mary's Hospital, for valuable suggestions in connection with the section dealing with questions appertaining to the organs of generation.

ARTHUR P. LUFF.

31 Weymouth Street, London, W.
1895.

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FORENSIC MEDICINE

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TOXICOLOGY

INTRODUCTION—MEDICAL EVIDENCE

CHAPTER I

Importance of knowledge of medical jurisprudence—Dying declarations—Inquests and procedure in the coroner's court—Medico-legal reports—Procedure in the magistrate's court—Procedure at the court of assize—Attendance of medical witnesses—Common and expert witnesses—Delivery of medical evidence—Fees of medical witnesses.

Medical Jurisprudence, or forensic medicine, or legal medicine, embraces every branch of the medical art and science. The importance of it to medical men is obvious, and cannot well be over-estimated. It may be reckoned with almost absolute certainty that a medical man, who has been engaged for any length of time in general practice, must on several occasions appear as a medical witness in one or other of the courts of law, and that upon his conduct on such public occasions, founded as it must be on his knowledge or want of knowledge of legal medicine, may depend the liberty and lives of some of his fellow-creatures.

When a diploma to practise medicine is taken, there is practically received with it a commission for the detection of

crime. For instance, at any time a medical man may be called to a case of poisoning or to a case of murder from stabbing or shooting; it is then impossible for him to avoid giving evidence, it is impossible for him to shift the responsibility to another; his public appearance in a court of law must follow, and upon his evidence the detection and punishment of crime may in great measure depend.

It is too frequently imagined by the medical student that a knowledge of medical jurisprudence can be spontaneously acquired when the critical moment for the application of such knowledge arrives. Nothing can be more misleading or inaccurate, and it is to be hoped that in the future more attention will be given to the study of the subject than has unfortunately been done in the past.

Medical jurisprudence is that science which teaches the application of every branch of medical knowledge to the purposes of the law. Its object is to furnish the instruction which can enable the medical man to adapt his knowledge and experience to emergencies, and to collate such medical facts and principles as, when arranged and given in evidence, may be necessary to enable a judge and jury to arrive at a just conclusion.

Medical Evidence

The duties of a medical jurist may be twofold: (1) to aid the law in fixing on the perpetrator of a crime; (2) to rescue an innocent person from a falsely imputed crime.

Medico-legal duties.—With regard to the various medico-legal duties of a practitioner and expert, detailed information will be given in the various sections of this work; but here it will be as well that advice should be given upon the following general points.

Necessity of careful observation.—When a medical man is summoned to a case which he may have reason to regard as suspicious, he should be careful to observe all details and minutiæ which are likely to have any bearing on the elucidation

of the case. A judge once pertinently remarked 'that a medical man when he sees a dead body should notice everything': for instance, any indications of a struggle; the position of the body; its relationship to other objects in the room or place where it is found, such as its position with regard to the door and to the various articles of furniture which may be present; and the nearness, or otherwise, to the body of any lethal weapon, or article from which poison may have been taken or administered. It is also advisable that a rough sketch showing the position of the body and its relationship to the various articles of furniture, &c. in the room should be made.

Clearness in language.—When giving evidence, simplicity of language and clearness of style should be zealously aimed at by the medical man. Highly technical language and vague statements should not be employed in the witness-box, and it is useless for medical men to think of sheltering themselves behind such language or statements, as criminal lawyers now acquire a considerable amount of medical knowledge in relation to questions of poisoning, abortion, wounds, &c. It should be borne in mind that it is the great aim and art of counsel who defend prisoners charged with murder or manslaughter, to endeavour to discover what the medical witness omitted to do, either during the treatment previous to death, or at the preliminary examination of the dead body, or at the post-mortem examination; and to attribute any omission either to professional ignorance or to professional bias.

Dying Declarations

It may sometimes be the necessary duty of medical men to take dying declarations. With the exception of dying declarations, the law requires all evidence to be given on oath, or what is equivalent to an oath. It is therefore necessary to carefully bear in mind the following three points which are required, so as to render such dying declarations without oath of equal value to evidence on oath.

1. The person must believe that he is in actual danger of death.

2. He must believe that his recovery is impossible.

3. To make the declaration admissible as evidence, death must have ensued.

The law assumes that no one would willingly leave this world telling an untruth during the last moments of life; and that if an accusation is made at so solemn a time, it is doubtless a sincere one. At the same time it should be remembered that the accusation may be sincere and yet not be true. Taylor and Stevenson cite a case of a dying woman in St. Thomas's Hospital who accused a man of assaulting her on Kennington Common; he was found guilty and executed; a year after his execution the real murderers were discovered, and the innocence of the executed man established.

This conviction of approaching death in the mind of the person making the dying declaration is absolutely necessary for its acceptance as evidence in a court of law; for in the case of *Jenkins* (Crown Cases Reserved, April 1869) a statement was rejected because the dying person, in using the expression 'I have no hope of my recovery,' requested that the words 'at present' should be added. In *Reg.* v. *Londesborough* (York Lent Assizes, 1871) Mr. Justice Brett declined to receive a statement because the evidence went no further than that the dying man said 'he thought he should not get better.'

Dying declarations are not available in civil but in criminal cases only, and they are only admissible in cases of homicide where the death of the person making the declaration is the subject of the charge. The declarations must relate to the actual circumstances of the death and to nothing else.

The same rules as to dying declarations hold good as to evidence on oath, viz.:—that a child of tender years must be proved to have had such religious knowledge as would have made the oath admissible. In the case of *Reg.* v. *Perkins* the dying declaration of a boy aged ten was received as evidence.

With regard to the duties of a medical man, when a dying person desires to make a declaration; if a magistrate or other legal functionary be present, his only duty is to give an opinion as to the state of the dying person's mind, *i.e.* whether he be in a sound mental condition or otherwise; but if no legal functionary be present, then it is the duty of the medical man to receive the declaration or confession; but he should be careful not to be officious in extracting information, and to simply write the identical words voluntarily uttered by the sick person; then, after reading the confession over to him, he should, if possible, get the dying person to sign it. In the event of the injuries being so severe and death so sudden that there is no time to write down the words of the dying man, then (whilst endeavouring to prolong life) it is the duty of the medical man to listen quietly to such statements as the dying person may make, and as soon as possible to commit them to writing. If other persons have been present and have heard these statements, this written account should be read over to them, and they should attest the veracity of the statements by attaching their signatures thereto. In such a case, it is allowable for word of mouth evidence of a dying declaration to be given, but greater weight is always attached to those statements that are taken down in writing in the presence of witnesses. Since, as was previously stated, in order to make the declarations admissible as evidence, death must have ensued, it follows that, if a person who has made a dying declaration recovers, the confession necessarily ceases to have any legal force. As has been already pointed out, the law makes an exception in the case of a dying person to the tendering of evidence on oath, since it assumes that, with the conviction of approaching death, he must feel himself under as solemn an obligation to tell the truth as if he were giving evidence on oath in the witness-box. If, however, after making a dying declaration a person should recover, then he resumes his normal condition with respect to the law, and must tender his evidence on oath in the witness-box.

Coroner's Court

The initiation of criminal proceedings in connection with a death in England and Ireland is generally in the court of the coroner; in Scotland, the office of coroner does not exist, but in place of it there is an officer named 'Procurator-Fiscal,' who is generally a qualified solicitor, and whose duty it is, in cases where a dead body is discovered, to obtain a written medical report of the cause of death from a duly qualified medical practitioner, if, in his opinion, such medical report is necessary for the due consideration of the case.

When death occurs from natural causes and under ordinary conditions, a certificate as to the cause of death is given by the medical man; such certificate, signed by a duly qualified and registered medical practitioner, together with a statement made by a person present at the time of death, or informed by some one who was present, is accepted by the registrar of deaths, who then issues a certificate authorising the burial of the deceased. A death-certificate should not be given by a medical man if he has any reason to doubt the cause of death being a natural one, or if the death has resulted either directly or indirectly from an accident or injury, when it becomes his duty to refer the case to the coroner; so that it will be seen that the whole responsibility rests upon the medical practitioner, since on the one hand he is compelled, under a penalty, to certify as to the cause of death, and, on the other hand, he may render himself liable, if certifying too hastily or upon insufficient grounds, to censure and, possibly, to legal proceedings.

If a medical man has fairly reasonable grounds for suspicion that the death of his patient did not result from natural causes, he should decline to give a certificate of the death, and should communicate with the coroner or the police authorities. With regard to the second reason for withholding a certificate of death—*i.e.* when death results from an accident or injury—there are two ways in which medical practitioners are not unfrequently brought into collision with the coroner; one is when death

takes place a long period after the occurrence of the accident, and especially when an intercurrent malady, such as an attack of pneumonia or of acute bronchitis, is the actual cause of death; in such a case the practitioner may naturally regard the disease, and not the accident, as the cause of death, and so may give a certificate accordingly; the second way in which medical practitioners may inadvertently come into collision with the coroner is by certifying the death of a patient who, while suffering from some chronic malady, meets with an accident not directly affecting life, such as a broken limb. Supposing that death occurs some weeks later, the medical man is prone to certify as if no accident had happened, especially as in such cases it may occur that pressure is put upon the medical attendant to induce him to give a certificate, and so to spare the family of the deceased the publicity of an inquest. In either of the instances that have been described, the coroner should, however, be informed, instead of a certificate being given.

Some coroners, on receiving information of the death of an individual, under suspicious circumstances, after satisfying themselves, as a rule, by hearsay evidence obtained through their officers, as to the absence of criminal causation or other reason for holding an inquest, intimate to the registrar of deaths that he may authorise the interment without an inquest being held. This is not strictly in accordance with the law, which provides that, 'except upon holding an inquest, no order, warrant, or other document for the burial of a body shall be given by the coroner' (50 & 51 Vict.).

It occasionally happens, in connection with some inquests, that the coroner fails to see the necessity of a post-mortem examination of the body being made; it is, however, extremely desirable, if not absolutely necessary in the interests of justice, that, with the exception of those cases of death resulting from machinery accidents and from railway accidents where the injuries are obviously the cause of death, a medical inspection of the body should be made in connection with all inquest cases. If, with the omission of the cases just referred to, there be

sufficient doubt as to the cause of death as to necessitate the holding of an inquest, there is also sufficient reason for a thorough inspection of the body at a post-mortem examination.

Coroners' inquests are sometimes considered too lightly by medical men. A fact, which is not generally known, should be carefully borne in mind—viz. that when evidence has been given before a coroner or magistrate, and the case is afterwards sent for trial, copies of the medical report and depositions given before the coroner or magistrate are usually placed in the hands of the counsel and the judge, so that the evidence given by a medical man before the coroner or magistrate may be compared word for word with that given at the trial. From ignorance on this point, it has not unfrequently happened, either from failure of memory, want of accurate observation, or carelessness in giving evidence at coroners' inquests, that medical witnesses have laid themselves open to severe censure by judges, either by stating matters differently at the trial, or by giving a different aspect or colouring to the facts; on such an occasion the prisoner's counsel is not slow to seize the opportunity of impressing upon the jury that a medical man who can give different accounts of the one transaction is not a person to be believed on either.

Inquests are generally held on information received from one or more of the following, viz. :—

1. A medical man communicates his suspicion to the coroner that the cause of death is not natural.

2. The beadle, or some other parish officer, informs the coroner of a sudden death, or of a death from a supposed unusual cause.

3. The registrar of deaths informs the coroner that no cause for death has been assigned, or that there has been rapid death after a short illness.

Under the present system, the coroner is empowered by the Medical Witnesses Act to issue an order for the attendance of any legally qualified practitioner, and an order may be issued to the medical man to previously make an inspection of the body

and to report the result. Both these orders a medical man is bound to obey; the summons issued is in the following form:—

'*Coroner's Inquest at the .... in the Parish of ... . in the County of .... upon the body of .... By virtue of this my Order as Coroner of .... you are required to appear before me and the Jury at the place aforesaid, on the .... day of .... 18 ...... at .... of the clock in the .... noon, to give evidence touching the cause of the death of the aforesaid person; and make, or assist in making, a post-mortem examination of the body (with an analysis of the contents of the stomach, if such be necessary), and report thereon at the said Inquest.*'

A fee of two guineas is the maximum allowed by the coroner for making a post-mortem examination, for giving evidence, and, if considered necessary, for making a chemical analysis of the contents of the stomach and of the other viscera.

With regard to the question of the analysis of the contents of the stomach and the viscera, medical practitioners usually decline to make a chemical analysis involving advanced knowledge of chemical manipulations and serious responsibility; a coroner has now only to make a representation to the Home Secretary, giving satisfactory reasons why the analysis is needed, and it is then conducted by one of the official analysts of the Home Office at no cost to the coroner, the expense being defrayed by the Government.

Medico-Legal Reports

Rules as to the points to which medical men should especially devote attention, either in the drawing up of medico-legal reports or in the delivery of evidence in the witness-box, will be given in detail in connection with the consideration of the subjects of poisoning, wounds, child-murder, &c., but, in this part of the book, it will be advisable to give some general instructions with regard to the drawing up of medico-legal reports.

I. As far as possible, technical and scientific terms should be avoided; for instance, instead of speaking of an 'hypertrophic liver,' speak of an 'enlarged liver'; instead of mentioning the 'duodenum,' mention the 'commencement of the small intestine'; instead of employing the terms 'gastritis' and 'enteritis,' use 'inflammation of the stomach' and 'inflammation of the intestine.'

II. Avoid the employment of terms and language which may be considered as exaggerated or wanting in definition; for instance, such terms as 'intense inflammation' and 'enormous dilatation' are relative terms, inasmuch as what may appear to be intense inflammation or enormous dilatation to one person may not appear to be so to another.

III. In medico-legal reports the facts should be kept distinct from the comments upon the facts; the facts are evidence, the comments are not necessarily so.

IV. The introduction of any hearsay statements, of statements made by others, or circumstances coming to the knowledge of the medical man through public rumour, should be carefully avoided.

The procedure in the coroner's court is very simple. Witnesses are examined on oath, their evidence is taken down, questions may then be put to them by any member of the jury, and, in the event of further proceedings being taken, they are bound under a pecuniary penalty to appear at the Superior Court to which the case is sent. If the evidence given at the inquest is incomplete, and if further evidence may be forthcoming, the coroner may then adjourn the inquest.

Magistrate's Court

Another preliminary court of enquiry is the Magistrate's Court; in this court the proceedings take the form of an investigation as to the culpability or non-culpability of a prisoner accused of some act of criminality or of negligence of a criminal nature. At this court, as it is now a question of guilt or innocence, the accused person must be present, and for the

same reason witnesses may be examined and cross-examined by counsel.

Non-important cases, such as simple assaults, may be dealt with summarily by the magistrate; but with more important cases, if the evidence of culpability is deemed sufficiently conclusive, the prisoner is committed for trial to a superior court, and the witnesses are bound over, under pecuniary liability, to appear there and give evidence.

Assizes

The next appearance of the accused person is at the Court of Assize. Before a case that has been sent up for trial from a lower court can be tried by a judge and jury, a preliminary enquiry is held before what is called the *Grand Jury*. The medical witness has to attend before the grand jury, to take the oath, and to give a general statement of what he knows of the matter or matters; he may have any questions put to him to elucidate the cause of death, but he is not submitted to cross-examination before the grand jury.

A true bill for murder or for manslaughter may be found by the grand jury, and the accused is then placed on trial before one of the judges of assize and a petty jury. Previous to the trial at the assizes a subpœna is issued to the medical witness, which subpœna he is bound to obey, but he is not bound to attend at the trial except upon a subpœna. If a medical witness is subpœnaed to give evidence as to matters of *fact*, then his fees are according to a fixed scale, particulars of which will be found at the end of this chapter. If he is subpœnaed to speak to matters of *opinion*, the question of fees is quite different, as he would then be occupying the position of an expert or skilled witness; in such a case, the fees are, as a rule, made a matter of private arrangement between the medical witness and the solicitor who serves the subpœna. If such an arrangement has not been effected, then a medical witness, before being sworn to deliver his evidence,

can in a civil case claim the payment of his fees. The solicitor serving the subpœna is not, however, liable for the fees, the principal for whom the solicitor is acting has generally been the person liable. As an illustration of the difference between the obligations of a medical witness in giving evidence as to matters of fact and in speaking as to matters of opinion, in the case of *Webb* v. *Page* (Carrington and Kirwan's Rep., p. 23), Maule J. ruled as follows : 'There is a distinction between the case of a man who sees a fact, and is called to prove it in a court of law, and that of a man who is selected by a party to give his opinion of a matter on which he is peculiarly conversant from the nature of his employment in life. The former is bound, as a matter of public duty, to speak to a fact which happens to have fallen within his own knowledge—without such testimony the course of justice must be stopped; the latter is under no such obligation, there is no such necessity for his evidence, and the party who selects him must pay him.'

With regard to the question as to whether a medical man subpœnaed to give evidence as to a matter of opinion is bound to attend upon such a subpœna, in *Betts* v. *Clifford* (Warwick Lent Ass., 1858) Lord Campbell stated, in answer to a question, that a scientific witness was not bound to attend upon being served with a subpœna, and that he ought not to be subpœnaed. If the witness knew any question of *fact*, he might be compelled to attend, but he could not be compelled to give his attendance to speak to matters of *opinion*. This expression of opinion, and the ruling by Maule J. previously mentioned, have been criticised by a barrister as follows : 'There is one reason why I should not advise any person in the position of a skilled witness totally to disregard a subpœna ; it is quite clear that, should such a person fail to attend the trial, no attachment could issue, even if he were called, as is usual, upon the subpœna, because the party subpœnaing him could not make the requisite affidavits that he was damnified by the witness's absence, and in what respect. But such party might bring an action for damages, and although he would recover none, he

might not only worry, but might even put the defendant to considerable expense, as "taxed costs" by no means include the entire costs in such cases. Although, therefore, I could not advise a total neglect of the subpœna, the safest course would be to obey it, and demand expenses before giving evidence; such expenses would be only those allowed for a professional witness (not special fees), but if the person, when so subpœnaed, were willing to run the risk of an action, he might safely absent himself without any fear of an attachment from the court for contempt' (Taylor and Stevenson[1]). The wiser course for a medical man who may not be acquainted with any facts of the case, and yet who receives a subpœna to attend and give evidence on matters involving the expression of opinion only, is to obey such a subpœna. In the event of separate subpœnas being received to attend trials at different assizes held at the same time, it is obvious that obedience to both of them is impossible; if one case should be a civil one and the other a criminal one, the criminal case should always take precedence of the civil one; if the subpœnas are for two criminal cases, that one which was first received should be the one to be first attended to, provided the days of the two cases clash.

Two classes of witnesses.—Medical men may be called either as common witnesses or as experts, or, what is more frequently the case, both as witnesses of fact and as experts.

I. A *common witness* is one who has to speak to matters of fact which have come within his personal knowledge; for instance, as to the injuries that may have been present upon a dead body or upon a living person whom he was called upon to treat.

II. The *skilled* or *expert witness* is called to give his opinion either on facts that may have come within his personal observation or that have been proved in evidence by others. The skilled witness should, in criminal cases, form his opinion on facts he has heard proved in the witness-box, and not on facts submitted to him in writing by the attorney, as it is necessary that he should be able to say that his opinions are

[1] *Med. Jurispr.*, vol. i.

based upon the evidence that he has himself heard given in the witness-box. For instance, Mr. Justice Hawkins, in the Staunton case, declined to take the expert evidence of a physician because he had not been present during the time that the witnesses had been giving evidence, but had based his opinions on the facts submitted to him in writing by the attorney. An expert witness is supposed to be specially skilled in the matters on which he is to testify. No one should venture to assume the position of an expert witness who has not devoted special attention to the matter under consideration, and who is, therefore, really able to enlighten the court and jury.

Answering questions.—It has occasionally happened that medical men have claimed a privilege not to answer certain questions which are put to them, on the ground that the matters involved in the questions have come to their knowledge through private and confidential communications made to them by patients. The law, however, concedes no special privilege of such a nature to members of the medical profession; in other words, there are no medical secrets. It is a matter which must be left entirely to the conscience and judgment of any medical man, as to whether he considers himself justified in disclosing facts in the witness-box which have come to his knowledge in his private relationship with a patient, and which, if disclosed, may seriously affect the happiness and domestic relations of a family; or whether, in the event of his declining to state the ground for refusing to answer such a question, he should risk the prospect of committal for contempt of court. A medical witness, like any other witness, is not bound to reply to any question if the answer is likely in any way to incriminate himself, since no man can be compelled to be a witness against himself. For instance, at a trial for murder by poison, in the course of cross-examination, the counsel for the prisoner asked the medical witness what remedy or antidote he had employed when he was first called to attend the deceased; he appealed to the judge to know whether he was bound to answer such a question as that, and the judge replied, 'Yes, unless you have

reason to believe that your antidote killed the deceased; in that case, you are not bound to answer it. The question was immediately answered' (Taylor and Stevenson[1]).

Before courts of assize barristers only can plead; whereas in a magistrate's court both barristers and solicitors can plead. At the Court of Assize, after opening the case, the counsel for the crown commences the examination of witnesses. Every medical witness is or may be subjected to three, and possibly four, sets of questions; these will now be considered in the order in which they may be put in court.

1. Examination-in-chief. 2. Cross-examination. 3. Re-examination. 4. Questions put by the judge.

I. **Examination-in-chief.**—The object is to bring out, by questions *which do not suggest their answers*, the facts relevant to the issue to be tried. These questions are put to the witness by the counsel representing the side on which the witness appears, on the strength of information contained in his brief, which consists of a full account of the case prepared by the solicitor who has charge of it.

With regard to written notes, a medical witness should not read his evidence, but, as the legal expression goes, he may refresh his memory from time to time by referring to written notes, provided always that these notes were made at the time of, or, if this was impracticable, immediately after, the occurrences to which they relate. The notes used must be the original notes, and not copies of them, and there should be no additions, the result of afterthought or of suggestions from others, made to the notes, if they are to be used in the witness-box. It is well to remember that, in the following cases, judges prefer evidence where the details have been committed to writing:—(i) Conversations with the prisoner. (ii) The results of a medical examination. (iii) The details of a post-mortem examination.

II. **Cross-examination.**—In this stage the counsel representing the opposite side to that on which the witness appears puts

[1] *Med. Jurispr.*, vol. i.

questions *which may suggest the answers in the strongest form*, in order to elicit any facts that may appear to be favourable to his client. These are called 'leading questions,' and the witness now occupies to the examining counsel the position of a hostile witness. It is during the cross-examination that any bias which may have influenced the mind of the witness, any flaws, omissions, and inconsistencies in evidence, are brought out. It is in this part of the examination that a medical man would be closely questioned as to the qualifications he possesses, the time that he has been engaged in practice, the special experience he may have had in reference to the subject matter of the trial, and the number of cases of a similar nature that he may have seen. To all these questions straightforward answers should be given.

III. **Re-examination.**—This is conducted on the part of the counsel by whom the witness was originally called, to clear up or explain any portion of evidence which may have been rendered obscure or doubtful by the cross-examination.

IV. **Questions put by the judge.**—Not unfrequently the judge may see fit to ask a witness certain questions that occur to him, in order to make clear certain points in his own mind, or for the information of the jury.

Rules for the Delivery of Medical Evidence

I. Questions put by the counsel on either side should receive direct answers, and there should be no perceptible difference in the manner of the medical witness when replying to a question put by the counsel for the prosecution or for the defence. It should be borne in mind that some counsel adopt the ingenious and misleading device of compressing two or three questions into one, so that a witness may unthinkingly answer the last, although the same answer may not be applicable to all, and yet, when too late to be recalled, it may be found that the answer has been so construed by the counsel in the defence. In such cases, when compound questions are put, the witness should

always ask for a severance of the questions, so that a separate answer may be given to each. With regard to giving evidence as to facts, the testimony of a medical witness must not be influenced by any consequences which may follow on their statement; but, in reference to matters of opinion, great care and consideration should be exercised, due regard being given to the possible influence which they may exercise on the fate of the prisoner. Previous to the trial, and, if possible, the night before it, the subject-matter of the evidence which the medical witness is about to give should be read through and carefully considered. Any standard works on the subject, and the opinions held and taught by others, should be referred to, as well as the precise grounds on which those opinions are held and taught. The facts concerning which the evidence is to be given should be arranged in the mind methodically and, as far as possible, chronologically. The witness should make himself quite clear as to dates and times, recalling, as far as possible, the day of the week as well as the month of any important event. It is also advisable, where the precise date or time is a matter of question, upon which important facts may turn, to be able to recall the circumstances that enable the witness to speak positively to the specific date or time. Where measurements, size, weight, &c., are matters of evidence, these should be carefully considered beforehand; in the witness-box it is advisable to adopt well-known English standards.

II. The replies to questions put by counsel should be concise, and, except where explanatory additions may be necessary, they should be confined to the terms of the question; excess of information in the answering of questions is generally the cause of a long and troublesome cross-examination.

It is well to remember that, when it is likely to have any bearing upon the elucidation of the case, an illustrative drawing is very useful. For instance, if a medical man has been called in to a case of murder or suspected murder, he should,

at the time, make a rough sketch showing the exact position of the body in the room or place where it was found, together with its relationship to articles of furniture, weapons, &c. Again, in describing injuries, such as cuts, stabs, bullet-wounds, &c., a sketch of the injury, particularly showing the exact position, size, and direction of the wound, is of great service in giving evidence.

III. The answers to questions put by counsel should be neither ambiguous, hesitating, nor evasive. A doubtful or hesitating answer, such as one commencing with 'I think,' or 'It might be,' is not sufficient for evidence. By previous consideration of the medical facts, a medical witness should have come to some conclusion on the subject, and should therefore be capable of giving a decided answer; if no conclusion has been arrived at upon the subject-matter of the case, then a medical man is scarcely in a position to give evidence.

IV. With reference to quotations from books, authorities should not be quoted in the witness-box. The medical witness is asked to give his opinion, and not to state whether such and such an author agrees or disagrees with that opinion; but an author may be quoted by counsel, who may quote or read a passage from his book, and the medical witness may be asked whether he agrees or differs with the opinion therein expressed. It is most important that the witness should take due care to see that the quotation is properly taken with the context. It is well neither to assent to, nor dissent from, the quotation until he has asked for the book, and has looked at the passages preceding and following it (for a counsel may put a different colouring upon the passage quoted, either by mistake or wilfully), since one sentence preceding or following the quotation read may frequently put an entirely new aspect upon the quotation itself.

Fees of Medical Witnesses

Coroner's Court.—A fee of one guinea is paid to a medical witness for attending to give evidence at a coroner's court when

no post-mortem examination is ordered. . An additional fee of one guinea is paid if a post-mortem examination has been ordered and made ; but this fee is only paid if the post-mortem examination has been ordered by the coroner in writing. There is no provision for a second fee for attendance at an adjourned inquest, but some coroners pay one guinea for every day's attendance.

When an inquest is held over the body of a person who has died in a public hospital, infirmary, public lunatic asylum, workhouse infirmary, or other medical institution supported either by endowments or by voluntary contributions, the medical officer of such institution is not entitled to any fee, and is bound to attend the coroner's inquest without fee ; but if the dead body of a person is brought into the public hospital, or other public institution, and the medical officer of that institution is summoned to give evidence, then he is entitled to the usual fee. The Coroners Act provides that immediately after the termination of the inquest the medical witness is to receive his fee.

Magistrate's Court.—If a medical witness resides within three miles, a fee of half a guinea is allowed for his attendance. At a greater distance, a fee of one guinea.

Court of Assize.—A medical witness is allowed one guinea a day, with two shillings for every night he is away from home, and second-class travelling expenses by rail; Sundays are not included.

Court of Probate and Divorce.—One guinea per day, if resident within five miles of the General Post Office; if at a greater distance, two or three guineas per day, with expenses for going and returning.

Court of Appeal.—A fee of one guinea per day is allowed if resident in London; two or three guineas if from a distance, with reasonable travelling expenses.

QUESTIONS CONNECTED WITH THE DEAD BODY

CHAPTER II

Signs of death—Cessation of heart's action—Cessation of respiration—Cooling of the body—Rigor mortis, or cadaveric rigidity—Production and disappearance of rigor mortis—Conditions affecting the time of commencement and duration of rigor mortis—Changes in the eyes—Changes in the skin.

The Signs of Death

A thorough knowledge of the changes which take place in the dead body at a recent, as well as at a remote, period must necessarily be of use in determining both the question as to whether death has actually taken place—a subject presenting, as a rule, no great amount of difficulty to the medical practitioner—or as to the period at which death occurred, a question which may be much more difficult to answer, and which may have an important bearing upon the demonstration of the guilt or innocence of an accused person.

Somatic death, or the death of the body as a whole, is defined as 'the cessation of the vital functions, and of the general renewal of tissue consequent on that cessation.' The signs of death are those enumerated under the following six headings:

1. The entire and continuous cessation of the heart's action.
2. The entire and continuous cessation of respiration.
3. The coldness of the body.
4. Rigor mortis, or cadaveric rigidity.
5. Changes in the eyes.
6. Changes in the skin.

Putrefaction, mummification, and the formation of adipocere, which are undoubted later signs of death, will be dealt with in the chapter on Putrefaction, &c.

THE ENTIRE AND CONTINUOUS CESSATION OF THE HEART'S ACTION

The cessation of circulation and respiration for any but a very brief interval of time is regarded as in itself sufficient to determine the reality of death. Life is not, however, incompatible with the brief apparent suspension of these two functions, but they must be speedily re-established, or death assuredly follows.

The rare possibility of a partial voluntary suspension of the heart's action should not be forgotten; the case of Colonel Townshend recorded by Cheyne [1] is an illustration of the extremely rare power possessed by an individual of partially suspending, by an act of volition, the action of the heart. Colonel Townshend, it appears, possessed the power, by a mere effort of will, of so suspending the action of the heart that he was able to pass into a profound sleep, or condition of stupor, in which no radial pulse could be felt, and no beating of the heart was perceptible by palpation. It must be remembered, however, that, at that time, the stethoscope was not invented, and that Colonel Townshend's heart must, in all probability, have been beating, although but feebly, during the time he was in the senseless condition, a period which usually extended over half an hour. A few hours after one of these experiments, however, the colonel died.

To detect the cessation of the heart's action, the stethoscope or the ear should be applied to the chest-wall, especially over the area of the superficial cardiac dulness, where the sounds are usually best heard; if no sound or movement of the heart is perceived during a period of five minutes, then death may be regarded as certain; but if any doubt should be entertained on

[1] *Treatise on Nervous Diseases.*

the subject, then it is advisable to wait for fifteen or thirty minutes, examining the heart from time to time with the stethoscope. Absence of pulsation in the radial artery at the wrist is no sign of the cessation of the heart's action.

The entire and continuous Cessation of Respiration

In order to observe whether respiration is occurring, the alternate movements of the chest and abdomen serve better for diagnostic purposes, and are more readily observed, than the movements of the chest alone; the visible cessation of these movements for a period of five minutes furnishes proof that the person is really dead. The stethoscope should also be employed to ascertain whether respiration has ceased or not, auscultation being conducted over the right and left second costal cartilages, to ascertain whether air is entering either bronchus. It is not necessary in this work to deal with the common tests frequently resorted to by the general public to discover whether respiration has ceased or not, such as holding a looking-glass over the mouth of the person to see whether moisture from the breath is deposited on its surface, or suspending a light feather or piece of wool near the mouth to see if movement occurs. These are, no doubt, fairly useful tests for inexperienced persons, but resort to them is quite unnecessary on the part of medical men.

Coldness of the Body

The temperature of the body, which during life is maintained at about 98·4° F., sinks after death until it finally becomes almost the same as that of the medium by which the body is surrounded. This loss of heat is a gradual and progressive one, and its progressive nature confers a special medico-legal importance upon post-mortem cooling, so that the temperature of a body may become, not merely a sign of death, but an indication of the time that the person has been dead. The time

taken to cool to the temperature of the air, or other medium by which the body may be surrounded, varies according to the following conditions.

I. **The condition of the body at the time of death.**—Bodies well covered with fat do not cool so rapidly as those which are emaciated; the bodies of very young or very old people cool more rapidly than the bodies of persons in the prime of life.

II. **The mode of death.**—After certain modes of death the body-heat may greatly exceed the normal; for instance, this has been observed after death from acute rheumatism, cholera, tetanus, strychnine poisoning, and some forms of apoplexy. Certain modes of death may cause a post-mortem elevation of temperature, or may retard the rate of cooling. If death is due to considerable hæmorrhage, such, for instance, as death from cut throat, the rate of cooling of the body may be considerably accelerated by the large loss of blood that has taken place.

III. **The amount of exposure and the temperature of the surrounding media.**—The rate of cooling of a body must necessarily be proportional to the difference in temperature between the body and the surrounding medium or media; a body which is well covered with clothes will necessarily cool more slowly than a body which is not covered; a body exposed to the open air, in cold weather, must necessarily cool more quickly than one in a closed room. The cooling of the body after death depends upon the following causes:—

1. The cessation of certain chemical processes in the body.

2. On radiation of heat—thus, a body will cool more rapidly in the open air than in a dwelling, more rapidly on the floor than in bed, more rapidly in a large apartment than in a small one.

3. On the conduction and convection of heat by the medium or substance in contact with the corpse: for instance, a body will cool more rapidly in water than in air, provided, of course,

the water is cold; more rapidly if naked than if covered with clothing; more rapidly in the case of a lean or emaciated body than in the case of a fat or corpulent one.

In the majority of cases the body is not cooled to the temperature of the air in less than fifteen to twenty hours; if, however, the conditions under which the body is exposed are favourable to rapid loss of heat, then a body may cool to the temperature of the air in from six to eight hours after death. It has been assumed by some writers on forensic medicine that the body cools at the rate of 1° Fahrenheit per hour, and that the average time taken for a body to cool down to the temperature of the air is about twenty-four hours. The rate of cooling of a body is not, however, uniform throughout, since it must depend on the difference of temperature between the body and its surrounding medium or media. For instance, in the case of a body exposed to the air, the rate of cooling directly after death may be at the rate of 4° or 5° or more per hour, whereas when the temperature of the body is approaching that of the air, the rate of cooling per hour is much slower, so that towards the end of the cooling the temperature may only fall a fraction of a degree per hour. In observations on the temperature of the dead body for medico-legal purposes, it is not advisable to depend on the sensation of warmth as communicated to the hand of the observer, but to employ a thermometer placed in the axilla to note the surface temperature, and one placed in the rectum to note the internal temperature.

Unusual retention of warmth after death.—Numerous cases have been recorded showing that the warmth of the human body may be retained in a greater or lesser degree for some time after death, and occasionally this exceptional retention of heat has given rise to the suspicion that the person may still be living. This retention of warmth in the body is especially likely to occur when death takes place at warm seasons of the year, and when the body remains in a room and is covered by materials which are bad conductors of heat, such as bed-clothes. The following case, illustrative of this, was reported in the 'Lancet'

some years since, and is quoted by Taylor and Stevenson.[1] 'A girl, who had retired to bed in apparently perfect health, was found on the following morning, as was supposed, dead. A surgeon who was called in pronounced her to be certainly dead, and stated that she had been probably dead for some hours. A post-mortem examination was ordered, in order that evidence as to the cause of death might be given at the inquest. The medical men making the examination found that the temperature of the body was not in the least diminished, although she must have been dead eight or ten hours.' Although, in this case, death had undoubtedly occurred, yet there were two causes influencing the retention of heat in the body; one was that the girl died suddenly while in a state of health and vigour, and the other was that the body remained in bed closely covered by the bed-clothes until the time of inspection.

Post-mortem rise of temperature.—After death from certain diseases the temperature of the body may actually rise for some time after the fatal event. The diseases in connection with which this post-mortem rise of temperature has been known to occur are cholera, yellow fever, rheumatic fever, small-pox, cerebro-spinal meningitis, strychnine poisoning, tetanus, and certain injuries to the brain. In connection with cholera the temperature has been known to rise, after death, as high as 113° F. This post-mortem rise of temperature is due to some chemical action taking place within the body, as the result of which there is the production of an excessive amount of heat.

In connection with the rise of temperature after death from cholera, some of the muscles not unfrequently undergo a contraction and subsequent relaxation. This phenomenon accounts for the authenticated observations that have been made of the bodies of corpses, after death from cholera, rising into the sitting posture; the movement is due to post-mortem contraction of the muscles which raise the trunk, the subse-

[1] *Med. Jurispr.*, vol. i.

quent falling back of which is due to the relaxation of those muscles.

Post-mortem sweating.—This occasionally occurs; a case is recorded [1] of a patient suffering from albuminuria, who, ten days before death, was seized with uræmic coma and left hemiplegia, which lasted to the end. Four injections of pilocarpine were given; the last (one-third of a grain) was administered three days before death, and produced a fair amount of sweating for twenty minutes. Forty-eight hours before death occurred profuse sweating broke out, and continued to the end. After death the body was washed and laid out, and when removed, four hours later, nothing unusual was noticed; sixteen hours after death the sheets in which the body was enveloped and the pillow upon which it was resting were found saturated, and the skin was moist and clammy; this continued for eight hours, *i.e.* until twenty-four hours after death. Post-mortem discolourations were well marked when the body was seen sixteen hours after death.

Rigor Mortis or Cadaveric Rigidity

By *rigor mortis* is meant the rigidity of the muscles that comes on after death, producing a stiffness of the limbs and joints. The muscular tissues of the body pass through three stages, and these stages furnish data which may be of extreme importance in determining, in any given case, the period that has elapsed since death occurred; they are—

First stage: *The stage of muscular flaccidity and contractility.*

Second stage: *The stage of rigor mortis or cadaveric rigidity.*

Third stage: *The stage of putrefaction.*

First stage: **The stage of muscular flaccidity and contractility.**—In the majority of cases seen after death there is a general relaxation of the whole muscular system; the lower jaw drops, the eyelids lose their tension, and the limbs become

[1] *The Lancet*, 1889.

soft and flabby. During this stage the muscles are not only flaccid, but they are capable of contracting under the influence of stimuli applied to them, such as the electric current, or smart blows from a stick, &c. So long as the muscles are capable of contracting in this way, they cannot be considered as actually dead; this stage generally lasts from three to ten hours, and must be regarded as the stage during which the muscles are dying, so that after the death of a human being, the muscles, in the majority of cases, pass through a dying period, lasting from three to ten hours, during which time they are capable of contracting under the influence of electrical or mechanical stimuli. Although this stage usually lasts from three to ten hours, yet, as will be mentioned later on, it may occasionally occupy only a few minutes, or even be apparently non-existent, so that, after death, the body may immediately pass into the condition of rigor mortis.

Second stage: **The stage of rigor mortis or cadaveric rigidity.**—Experiment shows that the seat of rigor mortis is in the muscles, for the rigidity disappears on their division. During the stage of rigor mortis the muscles retain the precise position they occupied at the time that rigidity supervened. The cause of the rigidity is the coagulation of the myosin, or muscle-fibrin, within the sarcolemma of the muscular fibres. With regard to the actual causation of the coagulation of the myosin, one view that has long been held is that it is effected by means of weak acids. Sarcolactic acid is being constantly formed during life, and as constantly removed from the muscles, and neutralised by the blood circulating through them; but after death, during the period of life that remains to the muscles, and while they are still flaccid and contractile, acid products continue to form and accumulate in the muscles, so that the muscles in a condition of rigor mortis have a strong acid reaction, due to the presence of sarcolactic acid, and possibly also of glycero-phosphoric acid. The rigidity of the muscles exists in two stages—an early stage and a later one, time simply determining the lapse of the former into the latter. In the

early stage of rigidity the myosin is probably only partially coagulated, and if, while the muscle is in this stage, fresh arterial blood is made to flow through its vessels, the semi-coagulated myosin is dissolved, and it is then found that the muscles are still irritable to electrical stimuli (Brown-Séquard). The later stage is due to the complete coagulation of the myosin, and when this stage is reached there is a permanent loss of irritability of the muscles to electrical or mechanical stimuli.

Tissot,[1] who has recently made some researches on cadaveric rigidity, has arrived at the following results:—(i) The rigid muscles can very often be electrically excited for a variable length of time after the onset of rigidity, and even when it is completely established. This persistence of excitability is almost constant in those cases in which *rigor mortis* has supervened rapidly. (ii) The rigid muscles which have lost their electrical excitability often preserve their mechanical excitability for a considerable period of time. (iii) The rigid muscles which have lost their electrical and mechanical excitability can still be excited to contraction by chemical reagents. (iv) Whilst the electrical excitability progressively diminishes, the excitability of the muscles to certain reagents increases, and reaches its maximum when that of electricity disappears, and at the moment when the muscle becomes rigid. (v) Tetanised and fatigued muscles present an exaggeration of sensibility to chemical reagents in the same manner as the rigid muscles. (vi) Contraction produced in rigid muscle by the action of some excitant is accompanied by the production of a 'current of action' in the muscle. It is also accompanied by the disengagement of heat, as in the contraction of normal muscle. (vii) Rigid muscles when exposed to the air absorb oxygen and give off carbonic acid.

In connection with the presence of sarcolactic acid in the muscle after death, Catherine Schipiloff[2] has formed an ingenious, though doubtful, theory of rigor mortis. She finds that

[1] *L'Union Médicale*, 1894.

[2] *Centralb. f. d. Med. Wissensch.*, 1882.

myosin can be precipitated from its saline solutions by weak acids without change, and that it is soluble in excess of acid; the same occurs in the body; by injecting small quantities of weak lactic or hydrochloric acid (0·1 to 0·25 per cent.) into the vessels of a recently killed frog, the muscles become rigid. This rigidity passes off by injecting stronger acid (0·3 to 0·5 per cent.). After rigor mortis, muscle contains more acid than either during rigor or before it sets in. The conclusion is therefore drawn that rigor mortis is due to the post-mortem development of acid, and its passing off is due to the further development of acid which redissolves the precipitated myosin. Halliburton believes that a ferment action is concerned in the production of myosin.

In addition to the hardness of the muscles produced by the state of rigor mortis, a slight amount of contraction takes place at its onset, and the muscles become to a slight extent shorter and thicker; the limbs, if left to themselves, stiffen in a position of partial flexion. As a matter of experiment, it is found that a muscle in the act of entering into rigor mortis can lift a weight, showing that actual contraction takes place. Coagulation of myosin alone does not account for all the phenomena in connection with the production of rigor mortis; the shortening of the muscles just referred to points to something more than mere stiffening. Some physiologists believe that cadaveric rigidity is accompanied by, if not actually the result of, a true muscular contraction.

It has been pointed out by Hermann[1] that the chemical processes taking place in muscular contraction during life, and in cadaveric rigidity, are analogous, with the exception of the formation of myosin from myosinogen. In both states carbon dioxide, sarcolactic and other acids are formed, heat is evolved, and the muscle current is reversed. Hermann considers that rigor mortis is an actual contraction of the muscles, produced by some form of stimulation, the contraction lasting longer, and passing off more slowly, than an ordinary muscular contraction.

[1] *Lehrbuch der Physiologie.*

Bierfreund[1] found, on dividing one sciatic nerve in recently-killed animals, that the advent of rigor mortis was always delayed on the injured side. Hemi-section of the spinal cord below the pyramidal decussation was also followed by delayed rigidity of the side on which the cord was cut. The effect of removal of the nerve influence on the process occurring in muscles after death, and prior to the advent of rigor mortis, was illustrated in an experiment made upon a dog, in which the cortex of the left half of the brain was stimulated during life, producing right-sided convulsions; the right half of the cervical cord was afterwards divided, and the animal was then killed. Ordinarily, the result of one-sided convulsions would be to hasten the advent of rigor mortis on the side on which they occur, but in this case the result of the removal of the nerve influences from the brain was that four hours and a half after death the limbs on the left side were in a condition of marked rigor mortis, whilst on the right side the limbs were almost as movable as at the time of death. Two hours later there was still considerable difference between the two sides. In the case of a man, who died forty-eight hours after an attack of cerebral hæmorrhage, there was found to be a delay in the occurrence of rigor mortis on the paralysed side. It is true that cadaveric rigidity does occur in paralysed limbs, but this and other recent observations indicate that its advent is delayed in the muscles of limbs so affected; this is important to bear in mind, since it was formerly stated that in hemiplegia there was no difference in the cadaveric rigidity of the two sides, provided the nutrition of the muscles was not affected.

Not only do the experiments of Bierfreund, just recorded, tend to prove that there is some actual relation between the production of rigor mortis and the nervous system, but Falk and Schroff have shown experimentally that irritation of the medulla can produce instantaneous rigor mortis. Moreover, in support of the more recent view that the nervous system does exercise some influence on the onset of rigor mortis, the

[1] *Arch. für die ges. Physiologie*, 1888.

observed phenomena in connection with the immediate onset of rigidity that occurs in some of the cases of instantaneous rigor, to which allusion will shortly be made, cannot be accounted for by the mere coagulation of myosin, but point to their production by some nervous or other influence. This subject, which will be further discussed later on, is of great importance to the medical jurist, since the tight closure of the fingers of a corpse around the handle of a weapon may be the sole evidence in favour of suicide, as against homicide.

Disappearance of rigor mortis.—Cadaveric rigidity generally disappears about the time that putrefaction commences, and the usual explanation of its disappearance is that it is due to the production of ammonia, resulting from putrefactive decomposition, which exerts a solvent action on the myosin, and so causes the stiffness of the muscles to disappear. It is true that myosin is soluble in an alkali such as ammonia, but there are many cases in which rigor mortis passes off before putrefaction sets in, and other cases in which rigor mortis persists after the onset of putrefaction. Hermann and Bierfreund state that cadaveric rigidity begins to disappear before any putrefactive changes take place; certainly, disappearance of rigidity has been observed in muscles retaining a strong acid reaction. It has been urged that to produce sufficient ammonia to dissolve the myosin, an advanced stage of putrefaction would be required, whereas rigidity generally passes off before any noticeable degree of putrefaction has taken place. Halliburton is of opinion that the disappearance of rigor mortis is due to the action of an unorganised ferment. All that can be stated with certainty is that cadaveric rigidity passes off about the time that putrefaction begins, but it is not by any means certain that putrefactive changes are necessary factors in bringing about its disappearance.

Time of commencement of rigor mortis.—As a rule, under ordinary conditions, rigor mortis commences about the third or fourth hour, and is complete about the sixth or seventh hour after death, so that it takes from two to three hours to affect all

the parts of the body. In the muscles of the face the rigidity is accompanied by a remarkable change of expression; the face is sallow, the jaws are fixed, the corners of the mouth are drawn downwards, the temples are sunk, and the brow is contracted—*facies Hippocratica.* By this rigidity of the facial muscles, the expression common to the person during life may undergo so complete a change, that identification may be rendered difficult, if not impossible, even by those who have known the person well.

Order in which the various regions and parts of the body are affected.—Rigor mortis generally begins in the muscles of the neck and lower jaw; from the neck it passes upwards to the muscles of the face, and downwards to the muscles of the upper extremities and trunk, and lastly attacks the muscles of the lower extremities. It passes off in the same order, so that the parts first entering into a condition of rigor mortis are the first to relax. The involuntary muscles are subject to rigor mortis as well as the voluntary, and, as a rule, it appears in them more rapidly.

The heart commonly enters into the condition of rigor mortis an hour after death, and remains in that condition for from twelve to thirty-six hours. It not only stiffens on entering into the condition of rigor mortis, but it also undergoes sufficient contraction to entirely alter the relative capacity of its cavities after death. When examined at this time, this abnormal condition has led to mistakes, the walls of the heart being described as thickened, and the cavities as diminished in size. This raises the important pathological question, as to whether the condition of the heart as found at a post-mortem examination, made at the time usually allowed to elapse after death, represents the condition of the heart at the time of death. For instance, in cases of death from syncope, the heart is usually found contracted, and the question is whether this condition represents the state of the heart at the time of death—*i.e.* whether death has taken place with the heart in systole, or whether the contraction was a post-mortem occurrence.

Some experiments made by Strassmann[1] are instructive in relation to this point. In a number of animals which had been killed by sudden paralysis of the heart, in some an examination of the heart was made immediately after death, and in others not until the next day. In those cases where the heart was examined immediately after death, the left ventricle was found to contain more blood than the right; but when the examination was made on the following day, the left ventricle was almost invariably found firmly contracted, and its contents expelled either into the left auricle or into the aorta. In all cases in which the heart was examined immediately after death, both ventricles were found in diastole, relaxed and filled with blood; in no case was the heart found to have stopped in systole. These facts indicate that the condition of the heart, at a post-mortem examination made some hours after death, gives no certain proof of its condition at the time of death. Most autopsies on the human subject are made after an interval of twenty-four or more hours after death, during which time the heart passes into a condition of rigor mortis, which may or may not have disappeared at the time of the examination; so that it is probably wrong to assume that the organ is then in the condition which existed at the time of death.

Conditions affecting the advent and duration of rigor mortis.—The average duration of rigor mortis is from twenty-four to thirty-six hours in summer, and from thirty-six to forty-eight hours in winter. The time at which it first appears and the period during which it lasts may be modified by various circumstances and conditions:—

(a) *Age.*—Rigor mortis occurs at an earlier period in the bodies of very young and old people than it does in those of middle-aged persons.

(b) *Condition and temperature of the surrounding medium.*—A dry and cold temperature will cause rigor mortis to persist for a long time, and, as a rule, delays its advent; whereas, if the air is warm and saturated with moisture, as in a tropical atmo-

[1] *Vierteljahrsschr. f. ger. Med.*, 1889.

sphere, rigor mortis will come on speedily and soon passes off. Taylor and Stevenson mention a case in which a body was exhumed three weeks after death, in the month of January. It was in a good state of preservation, and the limbs were so rigid that it required a great degree of force to bend them. In this case, no doubt, cold favoured the continuance of the rigidity. Bodies sunk in water, as a rule, speedily pass into a condition of rigor mortis, and retain their rigidity for a long time, so that the last struggles of life may be shown by the contracted state of the muscles, as occasioned by the struggles at the time of death. Taylor and Stevenson mention the case of a young man who, whilst skating, fell through the ice; he managed to keep his head and shoulders out of the water for some time by resting his arms upon the ice, but eventually sank. The body was recovered the next day, and the arms were then found stiffened in exactly the position in which they had rested upon the ice: the legs were extended, and the muscles of the front of the thighs were much contracted, as if they had been powerfully exerted in keeping him erect while hanging on to the ice.

(c) *The condition of the muscular system at the time of death.*—Whatever produces exhaustion of the muscles, such as violent exertion, accelerates the on-coming of rigor mortis. This accounts for the well-known fact that rigidity takes place rapidly in the dead bodies of animals that have been hunted to death after a long run, and also for the fact that the bodies of soldiers who are killed at the close of a long battle, after many hours of severe muscular exertion, become rigid almost immediately, whereas the bodies of soldiers killed in the early part of the battle enter slowly into the condition of rigor mortis. In this way the fact is accounted for that the bodies of soldiers are occasionally found stiffened in the actual attitude they were in at the time death occurred, so that they may be found kneeling with weapons firmly clenched in their hands, or even with the gun raised and kept at the shoulder. In these cases rigor mortis has occurred so rapidly that the muscles have at once passed into a condition of rigidity, without sufficient

time having elapsed for previous relaxation. The convulsant poisons, by their action on the muscles, cause rigor mortis to set in, as a rule, early after death, and in animals destroyed by strychnine it usually sets in with great rapidity.

Early rigor mortis has also been noticed in the bodies of human beings dying after violent convulsions produced by strychnine or by hydrophobia. Brown-Séquard found, in the case of a woman who died of hydrophobia after violent convulsions, that rigor mortis had set in within the first hour after death, and that it had ceased before the end of the tenth hour. Paltauf,[1] in connection with some experiments undertaken to show the causal connection between rigor mortis and deaths from certain poisons, found that curare always considerably delays the occurrence of rigor mortis, whereas strychnine, caffeine, picrotoxine, camphor, and the ammonium salts accelerate it. To study the influence of the nervous system at the time of occurrence of the rigor mortis produced by the last-mentioned poisons, Paltauf divided the nerves and spinal cord of the poisoned animal, and found that the more a muscle had been stimulated by the poison the sooner did rigor mortis occur.

(d) *Mode of death.*—As a rule, when death occurs from a lingering disease accompanied by great prostration, rigor mortis sets in quickly and disappears quickly. This may occur in death from phthisis, cholera, typhus fever, typhoid fever, hydrophobia, scurvy, and occasionally in chronic Bright's disease. Although, as a rule, rigor mortis, when it comes on speedily after death, disappears rapidly, yet it does not follow that such is always the case; for in some cases, where death has been preceded by powerful convulsions, rigidity may come on rapidly, and may continue for some days.

In cases of death from very lingering diseases, rigor mortis may last so short a time as to pass unnoticed, so that it has been stated that rigor mortis did not occur after death from those diseases. Such, however, is not the case; rigor mortis has occurred, but has lasted for a very brief period. John

[1] *Wiener Medicinische Presse*, 1892.

Hunter was of opinion that after death by lightning rigor mortis did not occur; this, however, is now known to be incorrect. Cases have been met with in which, after death by lightning, rigor mortis has been well marked and has persisted for some hours; but in other cases the persistence of rigor mortis has been so short that its existence has passed unnoticed. As a rule, in cases of speedy death occurring in persons of vigorous health, the onset of rigor mortis is delayed; to this, however, the peculiar condition known as instantaneous rigor must be regarded as an exception.

Instantaneous rigor or cadaveric spasm.—This is a peculiar and abnormal condition of rigor mortis, which occurs instantly after death, in which there is no interval of post-mortem muscular relaxation, so that the last living contractions of the muscles are actually prolonged into the rigidity of death. Dixon Mann [1] quotes the following examples of this strange phenomenon narrated by Seydel: 1. A man alighted from a railway carriage in order to procure refreshment. Whilst obtaining something to eat, he was disturbed by what he took to be the signal for departure, and ran across the line to his train, not noticing that the locomotive was being backed towards the first carriage; he probably saw the danger when it was too late, and in stooping to avoid it his head was caught between the buffers, and he was immediately killed. His arm was outstretched at the moment, and grasped in his hand were some provisions he had just obtained. Some hours afterwards the arm was still extended in the air, and the hand was firmly clasped on the food. 2. By the giving way of a bridge thirteen persons were suddenly precipitated into the water and drowned. Twelve hours after, when the bodies were recovered, in most of them the extremities were so firmly outstretched as to render it difficult to lay them in their coffins. 3. A man was standing on the ice, in the act of lighting a cigar; he fell through, and when the body was recovered, it was found in the upright posture, with the cigar and match between his fingers. 4. A

[1] *Forensic Med. and Toxicol.*, 1893.

man poisoned by carbonic oxide gas in his bedroom was found dead in a kneeling posture by the bedside.

Regnard and Loge,[1] in an account of some experiments that they made on the body of a recently decapitated criminal, state that two seconds after the head was severed from the trunk the jaws were firmly clenched; four minutes later the mouth was still firmly closed. The heart—ventricles and auricles—continued to beat rhythmically and strongly for twenty-five minutes after decapitation, and the auricles for forty minutes more. One hour after execution the heart was opened, when the left ventricle was found hard and contracted, the right remaining soft.

In the cases of instantaneous rigor quoted above, there has evidently been an absence of a period of muscular relaxation after death, and it is difficult to account for such instantaneous advent of rigor by the assumption that the condition is due to the coagulation of myosin alone. As the cause of the production of rigor mortis is still involved in obscurity, it will be sufficient here to refer to the fact that the experiments of Bierfreund, previously quoted, tend to show that the nervous system does exercise some influence on its production, and Falk and Schroff have demonstrated experimentally that irritation of the medulla produces instantaneous rigor mortis.

The phenomena indicative of instantaneous rigor or cadaveric spasm may be of immense importance in the interpretation of certain facts connected with the dead body. For instance, if a weapon, such as a pistol or razor, is found firmly grasped in the hand of a person, the condition is strongly confirmatory of suicide, since it is scarcely possible that such a condition could be produced by any arrangement of the fingers of the corpse around the weapon by a murderer after death. It is impossible for the fingers of a corpse, while still warm and supple, to be made to grasp a weapon in the same way that the hand had held it by strong muscular contraction at the moment of death; the attempts that have been made by murderers to produce

[1] *Le Progrès Médical*, 1887.

this appearance have always failed, the weapon in such cases being found lying loosely in the hand, and not grasped. On the other hand, it must of course be borne in mind that a weapon, although firmly grasped by a suicide at the time of the infliction of the wound, may either drop out of the hand or be found lying loosely in it, as a result of a muscular relaxation at the moment of death. So that, although a weapon found firmly grasped in the hand of a corpse is presumptive of suicide, on the other hand, the converse must not be assumed, viz.—that the mere fact of a weapon found loose in the hand is evidence of murder, since that condition may be due to muscular relaxation after a suicidal death. It has been asserted by Kussmaul, that the fact of a weapon being found firmly grasped in the hand of a dead person should not be taken as proof of suicide, since, as he states, if the fingers were closed around it in the stage of relaxation, they would in the state of rigidity tightly grasp the weapon. This objection, however, is a purely theoretical one, and on examination is obviously untenable, since it infers that murderers are acquainted with the medico-legal importance of causing the hand of a victim to grasp the weapon firmly, and also that they have a knowledge of the stages of rigor mortis, and would remain pressing the fingers of a corpse around a weapon until rigidity had set in with sufficient firmness to retain it in the hand.

Distinction of the rigidity of death from other forms of rigidity that may occur during life.—Rigidity during life from tetanus, apoplexy, catalepsy, syncope, or asphyxia has been stated to present symptoms somewhat resembling cadaveric rigidity. There are, however, these three striking differences:—

(i) If the rigidity occur in the course of any one of these diseases, the warmth of the body is preserved. (ii) In rigidity from disease, the whole of the body becomes equally rigid at the same moment, due to the occurrence of a general muscular spasm; whereas, in connection with rigor mortis, the rigidity commences in the muscles of the neck and lower jaw, and then gradually affects the other parts of the body. (iii) The distinc-

tion between muscular spasm in a living person and death rigidity is very simple. If a joint be forcibly bent, such as the arm at the elbow, the limb will, if in a state of spasm from disease, return to its original position when the bending force is removed, whereas if it be in a state of rigor mortis, it will not.

It should be remembered that rigor mortis in a new-born child does not indicate that it was necessarily born alive. It merely shows that it was recently living—*i.e.* it may have been living in the uterus or in the vagina, but have died before birth actually took place. For instance, a case has been recorded in which the body of a dead child, delivered after the operation of craniotomy, exhibited strong rigor mortis, but which obviously could not have been born alive. The point is an important one to remember in cases of alleged infanticide, since the mistake has been made of wrongly assuming that rigidity of the body is indicative of the child having been born alive.

Changes in the Eyes

These changes, as indicative of death, are mainly confined to the cornea and the iris.

I. **The cornea.**—Shortly after the occurrence of death the cornea generally loses its lustre and prominence, the globe gradually becomes collapsed and the surface wrinkled. The loss of lustre depends at first on the formation of a thin film of mucus over the surface; later on, the cornea tissue becomes opaque. The wrinkling and flattening of the cornea are due to the absorption of the aqueous humour which subsequently takes place. Although these changes in the cornea constitute a fair indication of death, it should be remembered that, in cases where blood has been forced towards the head, the eyes may preserve their brilliancy and prominence for some time after the occurrence of death. This may occur in cases of death from apoplexy, and after poisoning with carbonic acid gas, hydrocyanic acid, and cyanide of potassium. On the other hand, the film over

the cornea and the collapse of the globe have been observed in cases of malignant cholera several hours before the occurrence of death.

II. **The iris.**—The pupils dilate at the time of death, and do not respond to light. In addition, the iris is in a perfectly flaccid condition after death, as may be demonstrated by pressure with two fingers, one on either side of the pupil so as to compress the edges of the latter. If the person be dead, the pupil becomes irregular; whereas, if the person be still alive, the pupil retains its circular form, notwithstanding the compression. Although the pupils dilate at the time of death, and do not respond to light, Marshall [1] states that contraction subsequently takes place during a period varying from one to forty-eight hours after death; that atropine dilates the pupils after death, and that this effect may in some instances be produced after a lapse of four hours; and that eserine contracts the pupils, but not for so long after death.

In cases of catalepsy or trance simulating death, the pupils react to light.

Changes in the Skin

After death the skin becomes pallid and waxy-looking, from the absence of all circulation, and in some parts it becomes covered by livid discolourations or post-mortem stains. One of the most striking post-mortem changes in the skin is the almost entire loss of elasticity. In the living body, if any parts of the surface be compressed, the skin readily returns to its original form on removing the pressure; whereas, in connection with the skin of a corpse, the elastic recoil occurs but very slightly.

The so-called 'diaphanous test of death' is one that cannot be relied on. It consists in taking a hand of a supposed dead person, placing it before a strong artificial light, with the fingers extended and just touching each other, and then looking through the narrow spaces between the fingers to see if there be a scarlet line of light. The theory is that, if there be such a line

[1] *The Lancet*, 1885.

of scarlet colour, there is some circulation still in progress, and therefore evidence of vital action; whilst if there be no illumination, then the circulation has ceased and death has occurred. As showing the fallacy of this test, Haward[1] records a case of undoubted death, *rigor mortis* having commenced in the upper limbs, in which the scarlet line of light between the fingers was as distinct as it was in the hand of a living person subjected to the same experiment. Mason[2] mentions a case in which the diaphanous test was applied to the hands eighty hours after death by the aid of a lighted wax match; a bright scarlet colour was visible. On the other hand, Richardson[3] has reported an instance in which the test, applied to the hand of a lady who had simply fainted, gave no evidence of the red line.

[1] *The Lancet*, 1893. [2] *Ibid.*, 1894. [3] *Ibid.*, 1893.

CHAPTER III

The reality of death—Determination of the period at which death has taken place—Post-mortem stains or cadaveric lividities—Distinction of post-mortem stains from bruises.

The Reality of Death

The question of the reality of death is one that can only be raised in the period preceding the occurrence of putrefaction. It is one that should be considered in a work of this nature, since there is a prevailing idea, with some members of the public, that premature interment is occasionally the means of sending a living individual to the grave. Cases of alleged premature interment have however, upon close examination, proved to be nothing more than the delusions of superstitious or ignorant people, with a considerable amount of exaggeration added. Cases have certainly occurred in which persons in a condition of insensibility from concussion, coma, syncope, catalepsy, or exhaustion, have been pronounced dead by bystanders, not by medical men, simply because there were no outward signs of respiration or of circulation ; but in these cases the warmth of the body is retained, and the heart and lungs continue to act, although perhaps but feebly. There is no certain and simple sign which would be of use in guiding ignorant persons to decide on the reality of death soon after it has occurred, nor is it perhaps desirable that such should be discovered, as it would be always dangerous to leave the decision of such a question to ignorant people. It is better to strictly enforce the rule that nobody should be buried without a

medical certificate of death and its causes. To avoid the possibility of hasty interment of the living as dead, no burial should be allowed to take place until after the lapse of twenty-four hours, at least, from the time of the supposed death, and only then upon the certificate of a medical practitioner who has examined the body. If a proper interval be allowed to elapse after the supposed death, there should be no difficulty in deciding the question of the reality of death, prior to the commencement of putrefactive changes. The circumstances on which a medical man should rely as furnishing the best evidence of this are :—1. The absence of circulation and respiration. 2. The gradual cooling of the body; the extremities cooling first, and the trunk last. 3. The gradual supervention of rigor mortis.

Determination of the Period, prior to the commencement of Putrefaction, at which Death has taken place

A medical man, by careful observation of the state of a dead body, may, in cases of death from violence or suspected violence or poison, form a more or less correct estimate of the time at which death occurred, and, therefore, of the period at which violence was inflicted or poison taken. As there are, however, many factors which tend to modify the condition of the body at any one time, such as the age of the person, the mode of death, and the various circumstances that may have retarded or accelerated the cooling of the body, it is impossible for any hard-and-fast rules to be laid down, which can indicate that a given condition of the body must necessarily mean that a certain definite time has elapsed since death occurred.

In the determination of the period at which death took place, the striking of averages is not of much assistance, and in the following table the extreme limits of time compatible with a given condition of the body are presented. In giving an opinion as to the time that has elapsed since death occurred, it is for

the medical man, in any particular case, to draw his deductions from a correct observation of the state of the body when first seen, taken in conjunction with a careful estimation of all the causes which may have influenced or modified the various changes.

Conditions observable, prior to the commencement of Putrefaction, that indicate the Time at which Death occurred

Appearance and condition of the dead body	The inference is that death has occurred :
The body is more or less warm, and the voluntary muscles are relaxed	A few minutes to 20 hours
The body is perfectly cold throughout, and rigor mortis is well marked	10 hours to 3 days
The body is perfectly cold throughout; the limbs and trunk are free from rigor mortis; the muscles do not contract to electrical or mechanical stimuli; none of the putrefactive changes are observable	1 to 3 days (in hot summer weather); 3 to 8 days (in cool and cold weather)
The body shows the commencement of putrefaction, as indicated by a slight bluish-green discolouration of the abdomen around the umbilicus	6 to 12 days; but may appear in summer after 24 hours, and in winter may be delayed for 15 days

Post-mortem Stains

These changes are also known by the following names:—*Cadaveric lividities, cadaveric ecchymoses, cadaveric hypostases, post-mortem lividities* and *sugillations*. They are certain external changes that take place in a dead body before the commencement of putrefaction. On account of their colour and surface position, they have not unfrequently been mistaken for the effects of violence applied during life, and as serious mistakes may arise from such a confusion, it is very important that careful attention should be paid to them by medical men. The following

cases, mentioned by Taylor and Stevenson, illustrate the importance of such attention being paid to the subject. (1) A man was found one morning dying in a wood, and he died soon after discovery. On the previous day he had been seen to leave a public-house with two companions, all of them being at the time intoxicated and quarrelling with each other. Two surgeons deposed that they found the marks of numerous contusions all over the body, and upon this deposition the two companions of the deceased were committed and subsequently tried for murder. At the trial Bell and Fyfe proved that the apparent contusions were nothing else than the livid patches or hypostases which occur spontaneously on the dead body after many kinds of natural death; this led, of course, to the liberation of the accused persons. (2) A man named Keir and his mother were charged with the murder of the father of the man. The prisoners were condemned, but the only evidence of any weight against them was the appearance of a broad blue mark on the fore part of the neck, which the witnesses compared to that produced by strangulation. There was, however, great reason to believe, from their own description of it, that it was due to natural changes taking place after death.

It should be clearly understood that post-mortem stains are not the result of, nor are they in any way related to, putrefaction, although their effects have frequently been mistaken for it. They are certain after-death stains, which to the superficial observer resemble in their general appearance the effects of bruises or contusions. They occur both externally and internally (*i.e.* in connection with the various viscera) on the lowest and most dependent parts of the body: they show themselves generally during the four to twelve hours succeeding death; *i.e.* they appear during the act of cooling, whilst the body is still warm and whilst the blood is still fluid: occasionally they may be seen at an earlier period than the first-mentioned one. When once the blood is coagulated the formation of these post-mortem stains ceases.

Their production is explained in the two following ways:—

(i) Stagnation of blood in the capillaries. (ii) Gravitation of blood. During life the fluid blood is equally distributed throughout the circulatory system, but after death the blood ceases to circulate, and becomes subject to the laws of inert matter. It therefore gravitates to the lowest and most dependent parts of the body, and stagnating there produces congestion of the superficial capillaries, the walls of which have, by death, lost their contractility. It is the distension and congestion of the superficial capillaries by the blood which has gravitated to them that produces the post-mortem stains. These stains are necessarily found upon the most dependent parts of the body, provided those parts are not subjected to pressure. If the body be lying on its back, then the lobes of the ear, the shoulders, the lumbar region, the buttocks, and the posterior parts of the legs and arms will constitute the lower parts; if the body be in the prone position, then the face, chest, abdomen, and the anterior parts of the legs and arms will be the lower parts.

Post-mortem stains are of a purplish or dull-red character; they at first impart a mottled appearance to the skin, but, later on, the individual patches coalesce and form large areas of discolouration. These patches generally terminate abruptly in the white skin, so that they possess a well-defined though irregular outline. Their anatomical seat is the congested capillaries in the rete mucosum, just above the papillæ, and they are due, as previously stated, to simple congestion of the capillaries and not to sanguineous effusion.

The position of these post-mortem stains may vary, during the period that the blood remains fluid, if the body be disturbed; for instance, if post-mortem stains have made their appearance on the posterior part of the body as it is lying on its back, and if, while the blood is still fluid, the position of the body be reversed, the stains on the back part will disappear by gravitation of the still fluid blood to the parts that are now dependent, and fresh stains will be formed on the front of the

body. The post-mortem stains, however, become permanent in position when coagulation of the blood has occurred, and after this has taken place, no effect is produced upon them by altering the position of the body, nor are new stains formed; so that if the dead body has been moved, after coagulation of the blood has occurred, it may be found that the post-mortem stains will not correspond with its altered position, and so may afford evidence of the dead body having been interfered with some time after death. Although these stains appear on the most dependent parts, yet they do not occur on those portions of the body that are subjected to actual pressure. For instance, they would not be found on those parts where the body and the surface on which it is resting are in actual contact, since the superficial capillaries would be occluded by compression at the points of contact, and so the passage of fluid blood into them by gravitation would be prevented. Since external pressure may thus determine both the shape and the appearance of these post-mortem stains, it should be borne in mind that, if the body be closely wrapped in a sheet, the stains or lividities may in consequence be disposed in the form of a number of stripes over the surface of the body; this is due to the congestion of the capillaries taking place in the interstices of the folds, while the parts pressed remain white. In this way, an appearance may be given to the body, such as to lead one to suppose that the person must inevitably have been flogged during life, or a line round the neck, having the appearance of the mark of a cord, may be produced in a similar manner. A tight collar or neckerchief may act in this way, producing a mark round or partly round the neck, that may have the appearance of the mark left by a cord after death by strangulation.

The production of post-mortem stains in the internal organs should be carefully remembered, as the simple effects of gravitation can produce a livid or red appearance of the dependent portions of the stomach and intestines. This staining or hypostasis has been especially noticed in the small intestines, particu-

larly in those portions found in the cavity of the pelvis. This cadaveric congestion or hypostasis has been long known to exist in the lungs, where it affects, as a rule, the posterior fourth of each lung, and, consequently, there is a risk of attributing such a condition of the lungs to pathological causes. A similar cadaveric congestion of the stomach and intestines has not perhaps attracted such attention as the subject deserves, resembling as it does to some extent the effect produced by irritant poison. Such a mistake may be avoided by noting the absence of inflammatory exudation, and the fact that, on stretching out the opened intestines, the discolouration will be found absent in the divisions or folds lying between the discoloured portions. If the body has been lying on its back, the posterior halves of the kidneys are usually gorged with blood; the veins of the pia mater at the posterior part, the lateral and occipital sinuses, and the veins in the pia of the cord, are often found filled with blood.

The great importance of distinguishing these port-mortem stains from bruises inflicted during life is obvious. In the following table the distinguishing characters are given :—

Distinction of Bruises from Post-mortem Stains

—	Bruises produced during life	Post-mortem stains
Anatomical seat	Effusion of blood from ruptured vessels into the subcutaneous tissues and true skin	Congestion of the capillaries of the rete mucosum above the papillæ
Position	The seat of the injury	The dependent parts of the body not actually subjected to pressure
Appearance	Frequently have the shape of the instrument that inflicted the injury. The colour is not generally uniform, and the bruise is usually elevated	Irregular in shape, but with well-defined edges. The colour is uniformly purplish, and the stain is not elevated

Distinction of Bruises from Post-mortem Stains—*continued*

—	Bruises produced during life	Post-mortem stains
Results of incision	Effused blood, either in a clotted or fluid condition, is found	No effusion of blood is found
Changes by time	Zones of colour form around the edge, the centre always being the darkest. The colours are purple, violet, green, yellow, and lemon ; they are dependent on the different degrees of oxidation of the effused blood	No zones of colour around the edge. The purplish colour of the stain remains constant until putrefaction sets in

CHAPTER IV

Phenomena of putrefaction—Gases of putrefaction—Conditions affecting the production and rapidity of putrefaction—Mummification—Adipocere —Order in which putrefactive changes occur.

Putrefaction and Putrefactive Phenomena

In the term putrefaction a large number of chemical changes are included, which take place in all animal matter, as a result of the action of certain bacteria or micro-organisms. During the production of these changes offensive gases are evolved. The ultimate effect of putrefaction is the conversion of the complex organic compounds of the body into simple inorganic compounds, which are chiefly water, ammonia, and carbon dioxide. Putrefaction commences after the cessation of rigor mortis, and generally about the third day after death, when the limbs and trunk become soft and pliant, and yield a faint odour. The skin of the centre of the abdomen then assumes a pale green colour, which gradually deepens, and afterwards a similar discolouration slowly makes its appearance in other parts of the body. The order in which the colour-changes of putrefaction affect the various parts of the body is of medico-legal importance. The first putrefactive colour-change in bodies exposed to the air occurs, as previously stated, in the skin of the centre of the abdomen, and gradually spreads over the abdominal surface. A similar discolouration next appears on the surface of the chest, and then successively on the face, neck, legs, and lastly on the arms.

These colour-changes are due to an alteration in the colour-

ing matter of the blood that has infiltrated the skin and tissues, and are produced by the decomposition of hæmoglobin, as a result of the action of sulphuretted hydrogen and other gaseous products of putrefaction. The altered colouring matter permeates the tissues and colours them accordingly, and the distribution of the veins may be traced as dark lines. Putrefactive colour-changes make their appearance internally as well as externally. The signs of internal putrefaction first appear on the mucous membrane of the larynx and trachea.

Gases of putrefaction.—The gases produced by putrefactive changes are ammonia, sulphuretted hydrogen, methane or marsh gas, carbon dioxide, carbon monoxide, nitrogen, and phosphoretted hydrogen. Other gases or vapours are evolved, of the composition of which little is known at present, and to which the disgusting smells connected with the putrefaction of the body are doubtless mainly due. Some of these bad-smelling vapours are probably volatile ptomaines or animal alkaloids.

Distension of the body as the result of putrefaction.—From the development of gases of putrefaction and the resulting distension, the body swells and acquires a bloated appearance. This swelling takes place in all parts of the body, but since the gases of putrefaction meet with greater resistance to their escape in the tissues of the face, the effects of distension are especially noticeable there. In consequence of this the features become generally swollen, one or both eyes may be protruded, and the tongue forced out between the lips, while gaseous and frothy fluid may escape in bubbles from the mouth and nostrils. Protrusion of the eyes, as a result of putrefaction, should be carefully remembered, as such a condition has been wrongly denied by medical witnesses. It is true that, as previously described, the eyes of the dead become collapsed shortly after death, owing to the absorption of the aqueous humour; but as soon as the gases developed within the head are sufficient in quantity to force them forward, the eyeballs become more or less prominent.

This prominence of the eyes commences as a rule on the fourth to the eighth day after death. The extrusion of the tongue is partly due to swelling caused by the blood driven to the head, and partly to the development of gases in the tissues of the tongue. On account of its inability to be pushed backwards, it is pushed forwards as it enlarges, and also (should the head be in a dependent position) it may fall forward, so that from these two causes it may be found hanging out of the mouth after death. The medico-legal importance of this fact is, that the extrusion of the tongue and the prominence of the eyeballs have been wrongly considered at times by medical men as indicative only of death by strangulation; whereas, as just explained, these two phenomena may be the result of putrefaction alone. For the distinction of these conditions, see the chapter on Strangulation (vol. ii. p. 138).

As a result of the development of the gases of putrefaction, the body increases in size, and, in the cases of the bodies of children, this distension causes them to look considerably older than they really are. This is a matter of extreme importance, and should be carefully borne in mind when the precise age of an infant is in question. In a supposed case of infanticide, it has actually occurred in connection with the putrefied body of a still-born child, that, owing to the free development of gases, it has been mistaken for the body of a child a year old.

The gases of putrefaction accumulate under considerable pressure, and consequently various pressure effects may be produced. It is the pressure of the gases of putrefaction within the alimentary tract which accounts for the occasional escape of alimentary and fœcal matters from the outlets, phenomena which are apt to be regarded as vital ones by the uninitiated, and, consequently, are apt to cause alarm. On rare occasions, when women have died during labour and undelivered, the gases of putrefaction have accumulated with sufficient force to expel the contents of the uterus. This occurrence is known as post-mortem parturition. Aveling[1] considers that this phe-

[1] *Trans. Obstet. Soc. Lond.*, vol. xiv.

nomenon may occasionally be due to expulsion of the uterine contents by the contracting power of the uterus persisting after the death of the rest of the body ; he has recorded the occurrence of forty-four such cases, of thirty of which some particulars are given. Green [1] records the case of a pregnant woman at full time who died from puerperal convulsions. The body was laid out, about two hours after death, by two women, and at that time there was no birth, nor any signs of an impending birth. Fifty-three hours after death, when a post-mortem examination was about to be commenced, the dead body of a full-term child was found between the thighs of the mother. The uterus was inverted, and the fundus protruded from the vulva, having the placenta still attached to it; the perineum was ruptured to a considerable extent. Putrefactive changes were very advanced, the features were unrecognisable, and the abdomen was enormously distended with gases. On attempting to return the inverted uterus, it was immediately forced back again. On cutting into the abdomen, gas escaped with great force. It is quite probable that gaseous distension is the usual cause of the occasional movements of a corpse, such as its turning on the side after it has been laid in the coffin. Although the power of the gases generated by putrefaction may be very great, yet, on account of the elasticity of the tissues, it is seldom that a body bursts.

Blood displacements due to putrefaction.—Various blood displacements may be produced as the result of pressure exerted by the generation of the gases of putrefaction. In this way the occurrence of what is known as post-mortem hæmorrhage is explained. Post-mortem hæmorrhage may occur previous to the commencement of putrefaction if an open wound be present on the body ; it is then simply due to the liquid blood oozing from the wound, and afterwards coagulating. It may, and more frequently does, occur after the development of putrefaction, from the pressure of the gases, produced within the heart and blood-vessels, forcing the blood out of any wound that was made

[1] *The Lancet*, 1895.

before death. In this simple way is explained 'bleeding after death.' This post-mortem hæmorrhage would be facilitated by pressure, and hence arose the ancient but erroneous test of guilt —the touch of the murderer.

It should be borne in mind that occasionally the effects produced by blood displacement have caused considerable alarm, owing to the pallid face of a corpse suddenly becoming red and rosy. Taylor and Stevenson mention a case in which the face, three days after death, suddenly became red and suffused. This colour, however, disappears, and the original pallor quickly returns when exit is given to the gases confined within the thoracic and abdominal cavities.

During the putrefactive changes in the dead body, various discolourations take place in the mucous membrane of the stomach and intestines, which frequently closely simulate the effects of disease or poison. The mucous membrane may be tinted from a reddish-brown to a livid purple or even black colour. There are no invariable rules by which such putrefactive colour-changes can always be distinguished from those produced by disease or poison, but the two following observations are of great assistance in the discrimination of the redness that has taken place during life: (i) when the redness is seen soon after death; and (ii) when it is met with in parts not dependent.

Conditions requisite for the Establishment of Putrefaction

The primary conditions which are indispensable for the establishment of putrefaction in the dead body are:—

(i) A certain temperature. (ii) The presence of moisture. (iii) Access of air, which also means access of micro-organisms, such as the *bacterium termo*, which is a generic name for the micro-organisms of putrefaction and other allied bacteria, which doubtless are the active agents in determining the production of putrefactive changes.

In expressing an opinion as to the time at which death has taken place, the opinion being based upon a consideration of the putrefactive changes in the body, there is necessity for extreme caution and for a most careful consideration of the various circumstances and conditions which in the particular case may have either delayed or hastened putrefaction. There are many conditions which may greatly modify putrefaction, either hastening or delaying its advent. These conditions are the following :—

I. **Temperature.**—Putrefactive changes are produced most rapidly in bodies exposed to a temperature of from 70° to 100° F. (21° to 37° C.); whereas, on the other hand, putrefaction is wholly arrested at 32° F. (0° C.). A remarkable instance of the preservative power of cold has been given by Erman, who states that the body of Prince Menschikoff, a favourite of Peter the Great, exhumed after ninety-two years' burial in frozen soil in Siberia, had undergone hardly any change. Another wonderful instance of the preservative power of cold was shown in the discovery, in 1805, of the preserved body of an ancient elephant—the race of which was extinct before the historical period—in a mass of ice at the mouth of the river Lena, in Siberia.

As an instance of the way in which moderate warmth facilitates putrefaction, Taylor and Stevenson mention the case of a man who died in one of the wards of Guy's Hospital at night, and was not removed till nine hours and a half later, the temperature of the ward being between 60° and 65° F. When removed putrefaction had already commenced, and sixteen hours after death the condition was that which is generally assigned to dead bodies at a period of from six to twelve days after death. It is not uncommon for one day's exposure of a body in summer to effect a greater alteration than a week's exposure in winter. A warm room very materially promotes decomposition. Bodies buried in hot sand do not putrefy, but become mummified, so that it is only when warmth is associated with air and moisture that putrefaction is hastened.

II. **Moisture.**—Moisture accelerates putrefactive changes, in that it more effectually promotes contact between the air and the tissues. Putrefaction is also hastened if an excess of moisture be present in the tissues as the result of disease, such as dropsy. The viscera and tissues decompose at different times after death, according to the amount of moisture they contain; for instance, the brain putrefies rapidly, whilst the bones and nails decompose slowly. If there be sufficient water to allow of the entire submersion of the body, then putrefaction will be retarded on account of the prevention of access of air.

III. **Access of air.**—Air promotes putrefaction, partly by means of the oxygen contained in it, and partly because it acts as a carrier to the body of micro-organisms and bacteria, which produce, at all events, some of the chemical changes resulting in putrefaction. The body putrefies more rapidly if exposed to the air than if covered by water or if buried in the earth. Casper gives the following time-ratio of putrefaction in air, water, and earth. Given similar temperatures in the three cases, one week's exposure to air will correspond to two weeks' submersion in water, and to eight weeks' burial in the earth in the usual manner.

A naked body putrefies more rapidly than a clothed one, and decomposition is less rapid in those parts where the clothes fit tightly. Putrefaction is retarded by the prevention of access of air to the body, such as by enclosure in a leaden coffin. The effects of an entire exclusion of air in retarding the process of decomposition in a human body are shown in the burial of royal personages in leaden coffins, hermetically sealed, and afterwards enclosed in marble sarcophagi. When these have been opened, hundreds of years subsequently, the remains have been found in a remarkable state of preservation.

The condition of the air with respect to moisture and temperature will also have considerable influence on putrefactive changes. Moist and stagnant air promotes putrefaction, and moist, hot, stagnant air causes rapid putrefaction. Dry air retards putrefaction; air in motion also retards putrefaction.

A dry, cold air in rapid motion considerably retards putrefaction; and a dry, hot air in rapid motion also retards putrefaction, though not to the same extent as a cold air.

IV. **Age.**—The bodies of infants putrefy more quickly than those of adults, whereas the bodies of spare and old people putrefy slowly.

V. **Corpulence.**—Fat and flabby bodies putrefy more rapidly than lean bodies.

VI. **Mode of death.**—After death from exhausting diseases, such as typhoid fever, dropsy, and from septic diseases, such as pyæmia and septicæmia, putrefaction sets in, as a rule, early. After death from lightning and from asphyxia, early putrefaction generally takes place. Certain poisons, when they are actually the cause of death, may delay putrefaction, such as arsenic, chloride of zinc, and sulphuric acid; whereas, in cases of death from hydrocyanic acid and narcotic poisons, putrefaction is generally hastened. At the same time, it should be understood that the preservative property of a body like arsenic is not manifested in all cases of arsenical poisoning, so that it would be wrong to infer that a person has not died from the effects of arsenical poisoning because the viscera have undergone considerable putrefactive change in a given time.

VII. **Bruises, wounds, &c.**—Those parts affected by bruises, wounds, or extravasations of blood or serum, pass rapidly into a state of putrefaction. The fact of speedy decomposition setting in in parts so affected should be borne in mind, as otherwise it would be possible to make the mistake of considering that bruises, wounds, &c., when seen on a putrefied corpse, were considerably more aggravated in extent than was actually the case.

Internal Putrefactive Changes

The internal organs undergo putrefaction in a more or less definite order; the sequence of the putrefactive changes in them is partly due to the amount of moisture present, and partly

to the facility of access of air. Casper has formulated the order of internal putrefactive changes as follows:—

Organs that putrefy rapidly, placed in order

1. Larynx and trachea	5. Spleen
2. Brain of infant	6. Omentum and mesentery
3. Stomach	7. Liver
4. Intestines	8. Adult brain

Organs that putrefy slowly, placed in order

1. Heart	5. Œsophagus
2. Lungs	6. Pancreas
3. Kidneys	7. Diaphragm
4. Bladder	8. Blood-vessels
9. Uterus	

The influence of a moderately high temperature, and the stagnant atmosphere of a close room, in promoting rapid putrefactive changes in the dead body, should be borne in mind. The medico-legal importance of this will be made clear by the accounts of the following two cases which have been abstracted from Taylor and Stevenson's work on 'Medical Jurisprudence.'

At the trial of Mrs. Byrne in Dublin, in 1842, it was proved in evidence that the prisoner and her husband had retired to their bedroom, and about four days after the deceased had been last seen alive, and eight days after they had been in the room, the body of the husband was found dead on the bed. The wife, who had been all the time in the room, professed not to know that her husband was dead. The condition of the body was as follows:—The face and neck were black, the right eye protruded, the tongue projected about half an inch between the teeth, the ears were black, the lips swollen, and the fingers contracted. There was frothy liquid issuing from the mouth and nostrils, and living maggots were seen in those parts, and

the whole of the body was greatly swollen and discoloured. The body was found in this condition on July 9th. According to a statement made by the prisoner, the deceased died on July 8th, but she subsequently altered this statement and said that he died on the 9th, the day on which the body was discovered. From the state of decomposition of the body, two medical witnesses for the prosecution assigned a period of at least four or five days during which deceased must have been dead. On the other hand, a medical witness, called for the defence, deposed that he had seen a body as much decomposed twenty-four hours after death, and Geoheghan stated his belief that such changes might take place in from twenty-eight to thirty hours. Considering that the body was shut up in a close room at the hottest period of the year, and under conditions, therefore, most favourable to the acceleration of putrefaction, it is quite possible that the changes described took place within twenty hours of the time at which the body was discovered, although it must be admitted that this was an exceptional case of rapid decomposition.

At another trial, which took place in Ireland in 1863, somewhat similar questions were raised as to the early advent of putrefactive changes. On this occasion, the body of a woman was found dead in a room in an advanced state of putrefaction. The deceased and her lover—a soldier—had retired to a bedroom three days before the discovery of the woman's body, and had kept themselves there secluded. The medical evidence showed that when the deceased was first discovered on November 6th she was lying on her back in bed, the body being covered with clothes, the head and neck only being exposed, and there was a pillow lying loosely over the face; the upper part of the body was very much decomposed, and the skin of the face was so black that the features could not be recognised; the tongue was protruded and swollen; the lips were everted and blown up with gas; gases escaped from between the tongue and lips with a slight hissing noise; the abdomen was enormously distended with gas, and, at the lower part, was much

discoloured. On opening the abdominal cavity, the liver was found in a putrefied state. The body was found in this condition about sixty hours after the woman was last seen living. Considering that the weather was close and damp, and the body was shut up in a small room, there was ample time for the putrefactive changes described to have taken place, although such a degree of putrefaction is rarely seen until after the lapse of three or four days in warm damp weather.

On the other hand, the signs of putrefaction may be considerably delayed in cold weather. In 1824, a man named Desha was charged with the murder of Francis Baker. The deceased was last seen with the prisoner on November 2nd, and on November 8th, six days later, the body of the deceased was found, in a sheltered hollow, with the throat cut, and with wounds on the head and chest. From the nature of the wounds there was no doubt that the deceased had been murdered. The important medical point raised was whether the condition of the body was consistent with a suggestion of violent death, caused six days previously. When discovered, the body had undergone so little change that it was considered that the deceased must have been alive some days after the prisoner had been last seen in his company. The body, when first discovered, was a little stiff, and there was no sign of putrefaction about it; two days after its discovery, during which period it had been placed in a warm room, putrefaction set in. The fact of the body remaining in a sheltered spot during the month of November, without undergoing the usual changes of putrefaction, is quite consistent with death having occurred six days previously.

Accelerators of putrefaction.—The attempt occasionally made on the part of a murderer to accelerate the putrefaction of the body of the victim, by burying it in some supposed accelerator of putrefaction, generally fails. Lime, chloride of lime, sulphuric acid and lime together, have been employed, and the general result has been that either access of air to the body has been partially prevented, or the substance used has

exerted a disinfecting action, and so the result has been rather to preserve the body than to accelerate its putrefaction.

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Putrefaction in Earth

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It is impossible to give any certain opinion as to the length of time that a body has been buried in the earth. The reason is, that many conditions may modify the progress of putrefaction after burial, such as the character of the coffin and soil, the depth of the grave, the time that has elapsed before burial, and the cause of death; in addition, different bodies undergo putrefactive changes with very different degrees of rapidity, even when they have been under similar conditions. For instance, three bodies were buried at the same time, side by side, wrapped in cloth of the same texture, and in coffins of the same kind of wood. In connection with one of these bodies, it was found, at the end of nine months, that the abdominal walls had quite disappeared; in another, the disappearance of the abdominal walls did not take place until an interval of thirteen months from the time of burial; in the third one, at the end of twenty-three months the abdominal walls were almost entire. The usual period for the destruction of the soft parts, when bodies are buried in thin wooden coffins, may be taken at about ten years. Children's bones decay more rapidly than those of adults. The most indestructible parts of the body are the teeth, bones, and hair; these may be found in perfect preservation, after many years' burial, and may be of importance in throwing light on the age, sex, and identity of a person. It is occasionally the duty of a medical man to make a post-mortem examination on a putrid and offensive body, and this duty must not be shirked. In cases of suspected poisoning, no disinfectant should be added to the body previous to the performance of the post-mortem examination; but if the body is in so offensive a condition that a proper examination of it is almost impossible without previously rendering it less offensive, then the following mixture, recommended by Richardson, may be poured or sprayed

over the corpse, as it is obviously better, if any disinfectant be used, that its exact chemical composition should be known :—

Iodine	1 drachm.
Sulphuric acid	$\frac{1}{2}$ oz.
Absolute alcohol	1 oz.
Ether	10 oz.

This ethereal solution sinks quickly into the tissues of the corpse, the iodine acting as a deodoriser and antiseptic, while the sulphuric acid neutralises and fixes the alkaline products of decomposition.

Mummification

This change occurs under certain conditions in bodies, and consists in the evaporation of water from the soft parts, with the consequent conversion of the body into a brown, dry mass; to this desiccation of the soft parts of the body the term 'mummification' is applied. For its production a dry, warm air, with protection from moisture, is requisite; as, for example, when bodies are buried in hot, dry sand. The time required for the production of mummification varies with the conditions, but it has occurred in three months.

Adipocere

This substance derives its name from *adeps*, lard, and *cera*, wax, as it is a body intermediate in consistency between lard and wax. It is an impure ammoniacal soap, and consists of compounds of ammonia, with the following fatty acids:—oleic, stearic, and palmitic. During the putrefactive changes that occur in a corpse, the nitrogen of the nitrogenous bodies is largely converted into ammonia, which produces saponification of the fats of the body. Human fat is a mixture of olein, stearin, and palmitin; that is, a mixture of glyceryl oleate, glyceryl stearate, and glyceryl palmitate. When acted on by ammonia, saponification takes place, resulting in the formation of ammonium oleate, stearate, and palmitate, and glycerine. The mixture of these three ammonium soaps, in an impure state, constitutes adipocere.

If the corpse has lain in river water for a considerable period of time, or in a grave traversed by springs of water which are able to gain access to the body, then the adipocere found may be a lime soap; that is, a mixture of oleate, stearate, and palmitate of lime. This is due to the soluble lime-salts contained in the river or spring water decomposing the ammonium soaps, and converting them into lime soaps by substitution of lime for ammonia.

Conditions affecting the production of adipocere.—The parts first to undergo this change are those where fat is abundant, viz. the breasts, buttocks, and kidneys. The muscles may become converted into adipocere at later periods; the muscular tissue probably undergoes a fatty degeneration, which is followed by saponification of the fatty matters so produced. Adipocere is more rapidly formed in the bodies of children and young people than in those of adults; it is more rapidly formed in fat than in lean bodies. Its formation is favoured by complete immersion of the body in running water, and also by burial in a soil rich in ammonia and other putrefactive products, such as by burial in cesspool soil, or in a very crowded graveyard. Interment in deep graves also favours the formation of adipocere; no doubt from the body coming into contact with excess of moisture. If a body be buried either in sand or gravel, adipocere is rarely met with.

As regards the time required for its production, adipocere is not formed, to any considerable extent, under about three months' immersion in water (although traces have been found in from four to five weeks) or from six to twelve months' burial. If, however, the surrounding conditions are favourable to the development of ammonia, then the formation of adipocere may take place within a much shorter period. For instance, the body of a new-born child which had been thrown into the soil of a privy was found entirely saponified in six weeks' time. In hot climates the process may be extremely rapid. The following cases, reported by Mackenzie as occurring in Calcutta, are quoted by Dixon Mann.[1] (1) A male Hindoo

[1] *Forensic Med. and Toxicol.*, 1893.

was killed by the kick of a horse, and was buried the following day. Four days after burial the body was exhumed in order that an inquest might be held. It was in an advanced state of saponification externally, the heart and liver being also saponified. (2) A young Chinese woman, alleged to have died in child-birth, was buried; circumstances necessitated an inquest, and the body was exhumed seventy-six hours after interment, when it was found to be considerably saponified. These bodies were buried in a soft, porous soil, saturated with moisture, the temperature being high; the body last mentioned was enclosed in a wooden coffin. (3) Another case was that of a European sailor, who fell into the river Hooghly and was drowned; the body was recovered eight days and ten hours later. The external parts, the heart, liver, spleen, kidneys, stomach, intestines, and bladder, were saponified. (4) Another young European was drowned in the same river, his body being recovered seven days after. It was in an advanced state of saponification externally, and the lungs, heart, liver, kidneys, stomach, and intestines were also saponified; and what is very curious was, that the stomach contained undigested food—flesh and potatoes—of which the flesh was entirely saponified, the potatoes not being altered in the least. Other instances of early formation of adipocere are recorded as having occurred in India. In one case the body was saponified externally and internally in two days.

Order in which the external Phenomena of Putrefaction occur in Bodies exposed to the Air or Buried

From the description given in the previous part of this work, it has been made evident that the commencement of putrefaction may vary within the widest possible time-limits, according to the surrounding conditions. Moreover, variations in the degree of putrefaction, in the order of the putrefactive changes, and in the time taken by them, are very common, even when bodies are subjected to the same conditions.

Casper[1] mentions the case of two people, husband and wife, who were suffocated at the same time by carbonic oxide, and whose bodies were examined on the fourth day after death. The body of the man (who was thin) was putrid, whilst that of his wife (who was stout) was quite fresh. In addition, the time of the year, *i.e.* the temperature, may either considerably hasten the advent of the putrefactive processes or suspend it beyond the average period. It is therefore very important to remember that the following rules, laid down by Casper, should be regarded as average results only.

Appearance and condition of the dead body	The inference is that death has occurred :
Light green colour about centre of abdomen; eyeballs soft and yielding to external pressure	1 to 3 days
Green colour of abdomen intensified and spreading all over the body	3 to 5 days
Colour more intense. Face and neck reddish green. Ramifications of the subcutaneous veins very apparent. Gases beginning to distend the abdomen. Cornea fallen in and concave. Nails remaining firm	8 to 10 days
Colour dark-green, with brownish-red or brownish-black patches. Body bloated and appearing big from general development of gases. Features swollen, so as to render identification doubtful. Epidermis peeling off in patches and raised in blisters. Nails separate easily. Hair loose	14 to 21 days
Thorax and abdomen given way. Sutures of the skull given way from development of gases within. Viscera pulpy or melting away. Bones of extremities separated at joints	4 to 6 months

The infantile brain putrefies earlier than that of the adult on account of the absence of bony union of the skull-bones of the infant, which permits easy access of air to an organ prone to

[1] *Forensic Med.*, vol. i.

early decomposition. The order in which the various viscera putrefy is given on p. 58, from which it is seen that some organs putrefy relatively very late to others. For instance, the heart is found fairly fresh when putrefaction has already considerably advanced in the stomach and liver, and several months may be required to produce in the heart an equal degree of decomposition. The lungs generally show putrefactive changes about the same time as the heart. When the external signs of putrefaction are well advanced, the lungs may be found in good condition. Of all the soft organs of the body, the one which resists putrefaction the longest is the uterus, so that, even if the external parts of the body are destroyed by putrefactive changes, the sex of the corpse may be determined, as well as the occurrence, or not, of pregnancy or of recent delivery. It is especially in cases of alleged infanticide that a medical opinion is frequently required as to the probable date of the death of the child, and the question may be put to a medical witness, as to whether the condition of the body is consistent with the delivery of an accused woman at a particular period of time. It should be borne in mind that putrefaction, unless advanced to the final stage, does not entirely destroy marks of violence that has been attended by physical injury to parts, such as laceration of the skin, laceration of the muscles, and fracture of the trachea or larynx. In such cases, in spite of the decomposition that may exist in parts, a safe medical opinion can generally be formed.

With regard to the question as to whether putrefaction may commence in the body before death, partial putrefaction undoubtedly may, as gangrene is such a condition, but general putrefaction never can occur before death.

Putrefaction in Water

The process here takes place more slowly than in air, partly owing to the lower temperature, and partly to the prevention of free access of air.

When the colour-changes of putrefaction take place in bodies which have been immersed in water, the various parts are affected in a different order to that which takes place when putrefaction occurs in air. The discolouration commences in the face and neck, and then spreads successively over the shoulders, arms, chest, abdomen, and legs. This order is different to that which occurs when putrefaction takes place in the air, as will be seen by contrasting the two following lists :

Putrefaction in water : order of colour-changes in various parts of the body	Putrefaction in air : order of colour-changes in various parts of the body
1. Face and neck	1. Abdomen
2. Shoulders	2. Chest
3. Arms	3. Face
4. Chest	4. Legs
5. Abdomen	5. Shoulders
6. Legs	6. Arms

As gaseous putrefaction takes place in bodies immersed in water, the abdomen, the chest, and subcutaneous tissues become distended, and consequently the buoyancy of the body is increased, so that a time is at last reached when it will rise to the surface. To effect this, only a very slight expansion of the cavity of the abdomen is required, as the human body is only slightly heavier than an equal bulk of water. The position in which the human body floats is with either the abdomen or back lifted above the water, whilst the head and extremities are somewhat dependent below the water-level. In the case of females, it is invariably the abdomen that is lifted out of the water on account of the adipose tissue of the breast and abdomen. The period of time requisite for a body to rise to the surface varies, but it usually happens from the third to the eighth day after death from submersion. After the body has risen, the gases may be liberated and the body will then sink; gases may be again generated within the body, and it will again rise. For the data for determining the period at which death occurred in the drowned, see ii. p. 124.

It should be borne in mind that the dead body, when undergoing incipient decomposition, may become luminous or phosphorescent in the dark, just as dead fish may frequently exhibit such a phenomenon. This occasional phosphorescence accounts for many superstitions that formerly existed. Very occasionally it has been observed that the countenance of a dying person may also become luminous or phosphorescent in the dark, but this has only been very exceptionally observed, and then only in advanced cases of phthisis.

CHAPTER V

Causes of death—Common causes of sudden death—The modes of death—Syncope—Asphyxia—Coma.

PREVIOUS to the discussion of the various modes of death, it is advisable to refer to the common causes of *sudden death*. Sudden death may proceed from natural or violent causes, but putting on one side, for the present, death from violence and poisoning, the following are the common causes of sudden death.

COMMON CAUSES OF SUDDEN DEATH

I. *Heart disease*; especially in connection with aortic regurgitation, fatty heart, and rupture of the heart.

II. *Diseases of blood-vessels*; aneurism, thrombosis, embolism.

III. *Cerebral apoplexy*, or rupture of a blood-vessel on the surface of, or within, the brain.

IV. *Pulmonary apoplexy*, or rupture of a blood-vessel within the lungs.

V. *Perforation of the stomach or intestines by ulcers*, with consequent escape of food into the peritoneal cavity.

VI. *The sudden escape of blood into the peritoneal cavity*, from disease of the uterus, rupture of the uterus, &c.

VII. *Rupture of the bladder or other abdominal viscus.*

VIII. *Shock* from drinking large quantities of cold water when heated.

IX. *Mental emotions*, such as the sudden receipt of bad news, or violent fright causing sudden death.

X. *Foreign bodies* blocking the pharynx and causing obstruction of the glottis.

Templeman [1] describes two rare cases of sudden death. One occurred in a man, aged forty-three, from emotional inhibition of the heart; the heart was arrested in diastole, and there was entire absence of any pathological condition in any part of the body to account for death. The man had been in a condition of great excitement and passion a few minutes before death. The other case was that of a woman who died three or four minutes after receiving a severe blow on the pit of the stomach; the blow neither produced bruising nor rupture of any of the viscera; death was due to syncope, which was probably caused by an inhibitory influence on the splanchnics producing a sudden dilatation of the abdominal vessels, and consequently causing the withdrawal of such a large quantity of blood from the general circulation as to induce sudden anæmia of the central nervous system.

Among children the most frequent cause of sudden death is some form of laryngeal obstruction, generally *laryngismus stridulus*; another is the acute stage of pneumonia, in which they sometimes die suddenly, even with the disease unsuspected; these cases, according to Ogston, form a large proportion of the cases of death attributed to overlaying. Another cause is incomplete expansion of the lungs, children in this condition often dying suddenly a week or so after birth.

The Modes of Death

The continuance of life depends upon the proper action of the heart, lungs, and brain. When the stoppage of the heart's action is the primary cause of the fatal event, death occurs from **syncope**. When the primary cause of death is from affection of the lungs and interference with respiration, death

[1] *Edin. Med. Journ.*, 1893.

occurs from **asphyxia**. When the primary cause of death is disturbance of the functions of the brain, death occurs from **coma**.

TABULAR VIEW OF THE MODES OF DEATH

(A) **Syncope** (death beginning at the heart).

1. *Anæmia* (want of blood, but no want of heart power).
   (*a*) Loss of blood from wounds.
   (*b*) Hæmorrhages from the lungs, uterus, &c.
   (*c*) Extensive suppuration (indirect drain upon the blood).
2. *Asthenia* (want of heart power, but not necessarily want of blood).
   (*a*) Starvation.
   (*b*) Exhausting diseases, such as phthisis, cancer, &c.
   (*c*) Poisons exerting a depressing action on the heart.
   (*d*) Some injuries, such as a severe blow on the epigastrium.
   (*e*) Severe brain lesions.

(B) **Asphyxia** (death beginning at the lungs).

1. *Stoppage in action of the respiratory muscles.*
   (*a*) Exhaustion of the muscles from cold, &c.
   (*b*) Loss of nerve power from injury to the upper part of the spinal cord.
   (*c*) Mechanical pressure on the chest or abdomen.
   (*d*) Spasm from tetanus, strychnine poisoning, &c.
2. *Stoppage in action of the lungs.*—From entrance of air into the pleural cavity through a wound.
3. *Prevention of escape of air from, or of entry of air into, the lungs.*
   (*a*) Foreign bodies in the mouth, larynx, &c.
   (*b*) Submersion in water.
   (*c*) Suffocation, strangulation, hanging.
4. *Stoppage of blood supply to the lungs.*—From blocking of the pulmonary artery by a blood clot (embolism).

(C) **Coma** (death beginning at the brain).

(*a*) Pressure on the brain or medulla from apoplexy or compression.

(*b*) Blows on the head (concussion).

(*c*) Narcotic poisons, such as opium, &c.

(*d*) Certain mineral poisons.

(*e*) Blocking of a main artery of the brain by a clot or embolus.

(*f*) Certain kidney and liver diseases, such as uræmic poisoning, diabetic coma, &c.

The following table contains the most important symptoms preceding these forms of death. The recognition of these symptoms will be of use to a medical man suddenly called in to a case, such as a case of poisoning, by giving an indication of the line of treatment to be adopted.

Symptoms preceding the different Forms of Death

(A) Syncope.

1. *Anæmia.*

Symptoms:
1. Mortal paleness of cheek and lips.
2. Cold sweats.
3. Giddiness.
4. Slow, weak, and irregular pulse.
5. Insensibility towards the end.
6. Dilated pupils.
7. Convulsions generally supervene (from deficient supply of blood to the brain).

2. *Asthenia.*

Symptoms:
1. Hands, feet, and surface cold.
2. Fingers, lips, nose, and ears livid.
3. Pulse feeble and frequent.
4. Senses and intellect clear and active to the last.

(B) **Asphyxia.**

Symptoms:
1. Intense struggling for breath.
2. Giddiness.
3. Loss of consciousness.
4. Relaxation of sphincters.
5. General convulsions.

(C) **Coma.**

Symptoms:
1. Stupor.
2. Loss of sensibility and consciousness.
3. Breathing slow, irregular, and stertorous.

CHAPTER VI

Personal identity—Identity of the living or recently dead—Identity of mutilated remains—Identity of entire or incomplete skeletons—Distinction of human bones from those of the lower animals—Determination of sex in skeletons—Determination of age in skeletons—Determination of stature.

The question of personal identity may arise with regard to the living or the dead. Identity of the living may especially be required in connection with accusations of murder, assault, rape, and also in connection with a claimant to an estate—as occurred in the celebrated Tichborne case. The subject will be discussed in the following order:—

(1) *Identity in the case of a living person or of a body that has been dead a short time only.*

(2) *Identity in cases where mutilated remains have been discovered.*

(3) *Identity in cases where an entire or incomplete skeleton has been discovered.*

1. Identity in the case of a living person or of a body that has been dead a short time only.

The attention of the medical man should be directed to the detection and elucidation of the following points.

I. The *age* of the person or of the body.

II. The *sex*.

III. The evidence of the *occupation*, judging mainly from the condition of the hands.

IV. The *complexion*.

V. The general *type of face*.

VI. The *teeth*; the presence of false teeth should be care-

fully looked for, and also the presence or absence of individual teeth, as identity is frequently established by means of the teeth. If certain of the teeth are absent, examination should be made to see whether they have been removed recently or long before death.

VII. The *hair*; special examination should be made as to the colour, length, and texture of the hair, and also as to whether it has been dyed or bleached by some chemical agent.

VIII. The *nails*, especially noticing their length and any peculiarities of growth.

IX. The *stature* of the individual.

X. *Skin-marks*, and *birth-marks* or *mother's-marks*, such as nævi, moles, and tattoo-marks.

XI. *Scars and cicatrices of all kinds* should be carefully searched for. Birth-marks, such as nævi and moles, may have been removed by operation, but they always leave some scar behind, indicating removal. Any scars left from bleeding or cupping operations, and from vaccination, should also be carefully sought for.

XII. *Any deformities* that may be present should be noted.

XIII. *Injuries*, such as the existence of old fractures, should be carefully sought for. An old fracture is recognised by the callus or deposit around the bone at the point of union, and so may be readily distinguished from a fracture caused after death, such as by the spade of a digger.

XIV. In the case of a female, the existence or not of *pregnancy* should be ascertained.

XV. A careful description of the clothes and articles of jewellery found upon the body should be committed to writing.

2. **Identity in cases where mutilated remains have been discovered.**—This refers especially to those cases where the murderer divides the body of his victim into several parts, and distributes these in localities distant from each other, with the object of rendering identification difficult, if not impossible. Provided, however, the different parts of the body are recovered, and the soft parts still remain attached, there is, as a rule, very

little difficulty in settling the question as to whether the various parts belong to the same body.

The case of the woman Brown, murdered by Greenacre in 1837, illustrates the ease with which the question just referred to may be settled, provided the different parts of the body are recovered. The following account of the case is taken from Taylor and Stevenson.[1] The head, trunk, and limbs of the woman were scattered in widely distant parts of London. The limbs were not found until six weeks after the trunk, and then at a considerable distance, and under very different circumstances. In the examination of the trunk it was observed that the fifth cervical vertebra had been sawn through, leaving only about one-tenth of an inch of that bone. When the head was found it was observed that the fifth cervical vertebra had also been sawn through, leaving only the posterior spinous process. On comparing the head with the trunk they fitted exactly, even to the continuation of a superficial cut on the skin. On afterwards comparing the trunk with the legs, it was ascertained that the cut surfaces exactly corresponded. The thigh bones, remaining attached to the trunk, had been sawn through about an inch below the trochanters to about one-half of their thickness and then broken off. When the limbs were discovered six weeks afterwards, the portions of thigh bones found exactly corresponded in the marks produced by the saw, and in the portions broken. Not only were the parts of the body thus proved to belong to one and the same woman, but the individual was further identified by the peculiarity of the absence of the uterus.

The questions that require solution in connection with the identity of mutilated remains are :—(i) The *sex* of the deceased. (ii) The *age*. (iii) The *stature*. (iv) The presence of any physiological or pathological peculiarities. (v) The presence of *wounds* or *marks of violence* that may have any bearing on the mode of production of death. (vi) The general condition of the remains ; *i.e.* as to whether they may be parts of a dissected

[1] *Med. Jurispr.*, vol. i.

body, or as to whether they have been submitted to any chemical or other process for the purpose of preservation. (vii) The length of time that has probably elapsed since the death of the deceased person.

The following case, that of Harriet Lane, murdered by Henry Wainwright, indicates the mode of procedure in the examination of mutilated remains. The account is abstracted from Taylor and Stevenson.[1] On September 7th, 1874, the deceased woman went to a house belonging to Wainwright in Whitechapel Road and was never again seen alive. A year later Wainwright desired to remove the body of the woman, whom he had murdered and buried, to another hiding-place, and a man employed by Wainwright was arrested in the Borough having in his possession two packages containing human remains. On opening these, Larkin found one parcel to contain the trunk, and the other bundle to contain the two arms, two hands, two legs and feet connected, the two thighs with portions of the pelvis, and the head and neck. These parts were the remains of one human being, about five feet high, about twenty-five years old, and of a thin habit. The parts were in a stinking and decomposed state, but owing to the use of chloride of lime, employed with the view of destroying the body, certain parts had been well preserved. The cause of death was obvious, as two bullets were found in the brain and a third in a hair-pad at the back of the head. The identification was based partly on medical facts, and partly on the discovery of certain articles of dress in the grave from which the body had been removed. The direct proofs of identity were: (i) The *age*.—The age of the deceased was twenty-four; judging by the wisdom teeth, three of which had appeared, this corresponded with the age of the body. (ii) The *stature*.—It was inferred, but by no means proved, that the deceased was five feet and a quarter of an inch in height. The remains, when put together, represented a stature of four feet eleven and one-eighth inches. This makes about an inch difference,

[1] *Med. Jurispr.*, vol. i.

which was accounted for by one medical witness as a result of the shrinking of the intervertebral substances. (iii) The *colour of the hair.*—This is stated to have been slightly different, but the difference was consistent with the hair of the remains having been buried for a year in the earth, and exposed to the action of chloride of lime. (iv) A *scar.*—Harriet Lane was known to have received a burn many years before, which had left a scar or cicatrix on the right leg below the knee. On removing the adipocere the scar was found in the part indicated by the father of the deceased woman. It was distinctly puckered, and presented all the usual marks of a scar produced by a burn from a red-hot poker. (v) The *state of the uterus.*—The missing woman had had two children by Wainwright, the last having been born about nine months previous to her disappearance. The medical witnesses for the Crown—Bond and Larkin—after examining the uterus, came to the conclusion that the woman, whose remains were discovered, had borne a child.

3. **Identity in cases where an entire or incomplete skeleton has been discovered.**—The enquiries of the medical jurist must be directed to the elucidation of the following points :—

I. Whether the bones discovered are human bones or those of some animal.

II. If the bones are human, whether they are those of a male or female.

III. The determination, if possible, of the length of time that the bones have been in the grave.

IV. The probable age of the individual to whom the bones belonged.

V. The probable stature of the individual.

VI. If any of the bones exhibit fractures, whether those fractures had been produced during life, or by accident during exhumation.

VII. The presence or absence of fœtal remains.

In connection with this last-mentioned point it is well to bear in mind that undertakers frequently bury still-born children indiscriminately in coffins with adults.

Distinction of Human Bones from those of the Lower Animals

With regard to the distinction of human bones from those of animals, all that is commonly expected of a medical witness is to say whether a particular bone has formed part of a human skeleton or not. It is not necessary that he should say to what particular animal the bone may have belonged. Amongst the general public a considerable amount of ignorance prevails on these matters, the bones of cows, horses, sheep, and dogs being frequently mistaken for those of human beings. It is not necessary in this work to describe all the peculiarities of human bones, and the methods of distinguishing them from those of the lower animals, but the following are some of the well-marked differences which exist between the bones of men and of animals.

Skull.—The foramen magnum in all animals, except those of the ape tribe, is placed well at the posterior part of the base of the skull, and has its posterior edge turned upwards. In the ape tribe it is nearer to the centre of the base of the cranium, and so resembles the position of it in man. **Inter-maxillary bones.**—All animals, including those of the ape tribe, have these two bones in the face in addition to those found in man. On account of these bones holding the incisor teeth, they are sometimes called *ossa incisoria.* **Inferior maxilla.**—This in animals is destitute of the protuberance corresponding to the chin. **Clavicles.**—Most of the quadrupeds are destitute of clavicles. In the dog and cat there is a clavicular bone suspended in the muscles. **Pelvis.**—In animals the pelvis is always much elongated as compared with the pelvis of man.

Determination of Sex in Skeletons

The determination of the sex from an examination of the skeleton or of certain separate bones can, as a rule, only be made with certainty in subjects who have passed the age of puberty, since sexual differences in the skeleton do not become

very apparent until adult age has been reached. A full-grown bone of a female can, as a rule, be distinguished from the corresponding bone of a male by the ridges, depressions, and processes being less marked and defined, by the greater smoothness of the shafts, and by the articular surfaces being more flattened in female skeletons. The thorax is frequently flattened laterally, owing to the pressure of the stays. The thigh-bone of a female skeleton possesses a greater curve forwards at the upper part than does the thigh-bone of a male skeleton; and at the lower part it is turned more obliquely inwards than is the thigh-bone of a male. The neck of the femur in the female is more nearly at right angles with the shaft than in the male. The examination of the pelvis, however, furnishes the most satisfactory method for the determination of the sex in an adult skeleton. In the male the bones are thicker and stronger, and the muscular eminences and impressions on their surfaces are more strongly marked. The male pelvis is altogether more massive, its cavity is deeper and narrower, and the obturator foramen is of larger size. In the female the bones are lighter and more expanded, the muscular impressions on their surfaces are only slightly marked, and the pelvis generally is less massive in structure. The iliac fossæ are broad, and the spines of the ilia are more widely separated, hence the great prominence of the hips. The inlet and outlet are larger, the cavity is more capacious, and the spines of the ischia project less into it. The promontory is less projecting, the sacrum is wider and less curved, and the coccyx more movable. The arch of the pubes is wider, and its edges more everted. The tuberosities of the ischia, and the acetabula, are wider apart. In the fœtus, and for several years after birth, the pelvis is small in proportion to that of the adult; the cavity is deep, and the projection of the sacro-vertebral angle less marked. The antero-posterior and transverse diameters are nearly equal. About puberty, the pelvis in both sexes presents the general characters of the adult male pelvis, but after puberty it acquires its proper sexual characters (Gray).

Determination of Age in Skeletons

The examination of the jaws and teeth, together with the progress of ossification in the skeleton, and the general surface appearances of the bones, are the sources upon which mainly to rely as furnishing evidence of age in skeletons. At birth the angle formed by the ramus and the body of the lower jaw is obtuse, being equal to about 140°. The body of the jaw is shallow and semicircular in form, but as adult age is reached the body grows posteriorly and gradually changes its form to a horse-shoe shape, while at the same time it becomes deeper and thicker. The ramus lengthens, and the angle formed by the ramus and the body of the jaw becomes less obtuse, until it approaches a right angle. In old age the teeth are generally lost, and the alveolar portion of the jaw atrophies, so that the body of the jaw becomes again shallow, and the angle formed by the body with the ramus becomes again obtuse. The body of the infantile jaw is mainly alveolar, whereas the body of the jaw at old age is entirely basal, as can be demonstrated by the relative position of the mental foramen in the two cases; in the infantile jaw the foramen is low down, whereas in the jaw of old age it is near the upper border.

Examination of the Teeth

Periods of the eruption of the temporary or milk teeth.—These appear in the following order.—

Sixth or seventh month . . .	Lower central incisors
Eighth ,, ...	Upper central incisors
Seventh to ninth ,, ...	Upper lateral incisors
Tenth to twelfth ,, ...	Lower lateral incisors
Fourteenth ,, ...	Four first or anterior molars
Seventeenth to eighteenth month .	Four canines
Twenty-second to twenty-fourth month	Four second or posterior molars

The temporary or milk teeth are twenty in number; their arrangement is shown in fig. 1.

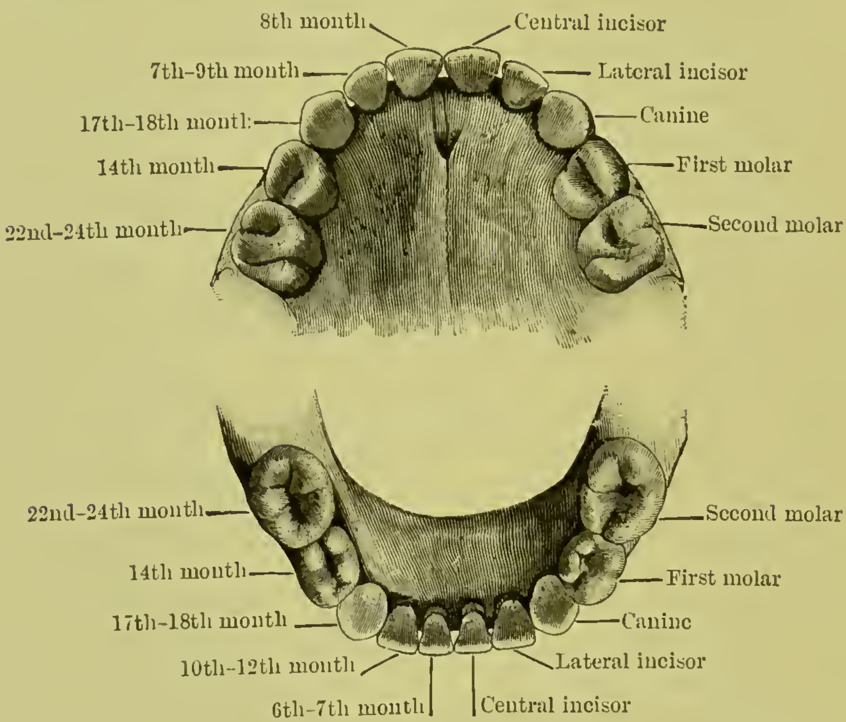


FIG. 1.—PERIODS OF ERUPTION OF THE TEMPORARY TEETH
(Smale and Colyer's 'Diseases of the Teeth')

Periods of eruption of the permanent teeth.—These appear in the following order:—

Sixth or seventh year . .	Four anterior or first molars
Seventh or eighth ,, . .	Four central incisors
Ninth year	Four lateral incisors
Tenth ,, ...	Four anterior bicuspids or pre-molars
Eleventh to fifteenth year .	Four posterior bicuspids or pre-molars
Eleventh to thirteenth ,, .	Four canines
Thirteenth to sixteenth year	Four second molars
Eighteenth to thirtieth ,,	The third or last molars or wisdom teeth

The permanent teeth are thirty-two in number; their arrangement is shown in fig. 2.

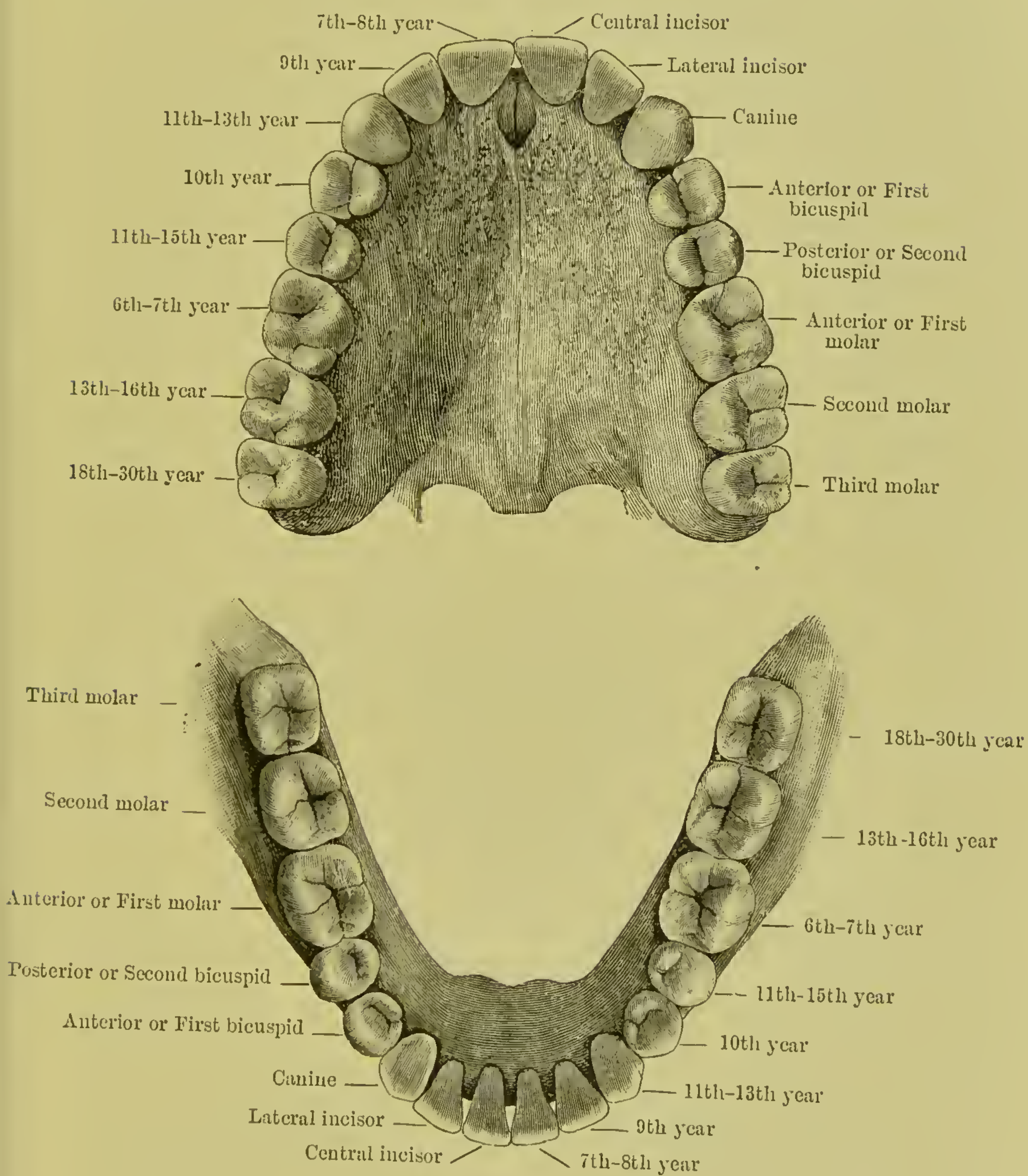


FIG. 2.—PERIODS OF ERUPTION OF THE PERMANENT TEETH (Smale and Colyer's 'Diseases of the Teeth')

The following tables are arranged for the purpose of determining the probable age of a body or skeleton from the progress of ossification; they are taken from Quain's 'Osteology' and Dixon Mann's 'Forensic Medicine.'

TABLE SHOWING THE PERIODS AT WHICH POINTS OF OSSIFICATION APPEAR AFTER BIRTH

Years of life	Bones in which the ossification-points occur
1st . . .	Fourth piece of the body of the sternum
,,	Coracoid process of the scapula
,,	Head of the humerus
,,	Os magnum (carpus)
,,	Head of femur
,,	Upper end of tibia
,,	External cuneiform (tarsus)
2nd . . .	Lower end of radius
,,	Unciform (carpus)
,,	Lower end of tibia
,,	Lower end of fibula
3rd . . .	Great tuberosity of humerus
,,	Patella
,,	Internal cuneiform (tarsus)
3rd to 4th .	Upper end of fibula
4th . . .	Great trochanter (femur)
,,	Middle cuneiform (tarsus)
4th to 5th .	Scaphoid (tarsus)
,, ,,	Lower end of ulna
5th . . .	Lesser tuberosity (humerus)
,,	Internal condyle (humerus)
,,	Trapezium and semi-lunar (carpus)
5th to 6th .	Upper end of radius
6th . . .	Scaphoid (carpus)
7th . . .	Trapezoid (carpus)
10th . .	Upper end of ulna
12th . .	Pisiform (carpus)
13th to 14th .	External condyle (humerus)
,, ,,	Small trochanter (femur)

After a person has reached full adult age, the examination of the bones will reveal the following conditions: (i) Union of all the epiphyses of the bones. (ii) Firmness and solidity of the bones. (iii) Rough surfaces for the attachments of muscles. (iv) Well-marked processes, grooves, and foramina.

TABLE SHOWING THE PERIODS OF UNION OF EPIPHYSES WITH SHAFTS OF BONES, AND OF BONES WITH EACH OTHER

Years of life	Epiphyses of bones
1st or 2nd .	Symphysis of lower jaw
2nd . .	Frontal suture; unites from below upwards (may persist)
,,	Anterior fontanelle filled up
7th or 8th .	Rami of ischium and pubis
17th . .	Epiphysis of upper end of ulna
,,	,, ,, small trochanter (femur)
17th to 18th	,, ,, condyles (humerus)
,, ,,	,, ,, upper end of radius
18th . .	,, ,, great trochanter (femur)
,,	,, ,, lower end of tibia
,,	Lower sacral vertebræ
,,	Portions of acetabulum united
19th . .	Epiphysis of head of the femur
20th . .	,, ,, ,, ,, humerus
,,	,, ,, lower end of radius
,,	,, ,, ,, ,, ulna
21st . .	,, ,, upper end of tibia
,,	,, ,, lower end of fibula
24th . .	,, ,, upper end of fibula
25th . .	Second and third pieces of sternum
,,	First and second sacral vertebræ
,,	Epiphysis of clavicle
,,	,, ,, lower end of femur
40th . .	Manubrium with body of sternum

The bones of an old person present the following peculiarities: (i) Parts which are cartilaginous in adults, such as the costal cartilages, thyroid cartilages, and trachea, become more or less ossified. (ii) The bones of the cranium become much thinner, and the sutures disappear first on the inside of the skull, and later on the outside. (iii) The teeth have dropped away from the jaws or the crowns are worn away to the sockets. (iv) The alveolar cavities sometimes quite disappear, the lower jaw becoming, more or less, a rounded bone.

DETERMINATION OF STATURE

The average stature of Englishmen is five feet seven inches. In determining the stature from a measurement of an entire

skeleton, it is customary to add from an inch to an inch and a half for the thickness of the soft parts. If the bones are entirely disarticulated, they should be laid out in their natural order and in juxtaposition, and then a measurement should be made. With regard to the endeavours that have occasionally been made to ascertain the stature of a skeleton from the measurement of one of the long bones, very unsatisfactory results are, as a rule, obtained, as there is no constant relation between the length of any one bone and the stature of a person. From the bones of the arm, or from an entire arm, it is however possible to obtain approximately the stature of a person. There is, as a rule, a remarkable approximation between the stature of an individual and the transverse measurement taken between the extreme ends of the middle fingers, the arms being held outstretched in a horizontal position. If, therefore, only one arm is obtainable, by doubling the length of the arm, and adding twelve inches for the two clavicles and one inch and a half for the width of the sternum, a very close approximation to the stature of the individual is obtained.

With regard to the determination of age from the stature of a body, nothing very definite or trustworthy can be made out, since there are great differences in the rapidity of growth in children either of the same sex or of different sexes.

CHAPTER VII

Post-mortem examination for medico-legal purposes—External examination—Internal examination—Instruments—Preservation of viscera for analysis—Exhumation.

Method of Making a Post-mortem Examination for Medico-legal Purposes

The examination should be made in daylight, as some colour-changes are very apt to escape detection in artificial light. It is desirable that two medical practitioners should conjointly make the post-mortem examination, and that the results should be recorded by one at the dictation of the other who is actually conducting the examination; as soon as the necropsy is finished the operator should read over the notes, which should then be signed by both medical men.

Before the clothes are removed from the body, the hands should be inspected to see whether anything is grasped in them, such as portions of hair or clothing, or whether they are stained with blood, or blackened by gunpowder, or have cuts or other injuries upon them. The clothes are then to be removed, and any cuts or other injuries they may have sustained should be carefully compared with the underlying surface of the body.

External examination of the body.—Notes should be made of the temperature of the body, the degree of rigor mortis, the presence or absence of putrefaction, the height, weight (if dealing with the body of an infant, or in a case of suspected starvation), sex, apparent age, amount of muscle and fat, colour of the skin, scars, moles, nævi, and other marks which may serve for purposes of identification, the number and pecu-

liarities of the teeth, the colour of the eyes and hair, and the presence or absence of foreign bodies in the mouth, nostrils, pharynx, anus, or vagina. Blood-stains, contusions, and wounds should be carefully looked for; post-mortem stains must not be mistaken for contusions (see p. 48). The extent of wounds and contusions should be investigated by dissection, not by probing. The skin surrounding a gun-shot wound should be examined for blackening or tattooing. The neck should be examined for marks of strangulation. If the top of the head has sustained any injuries, the hair must be shaved off in order to examine their superficial extent. If a wound be found on the body, and a weapon be discovered, the examiner should form an opinion as to whether the injury could have been inflicted by such a weapon, and also as to whether the wound could have been self-inflicted or not. In the case of the examination of a female body, the condition of the external genitals and of the hymen should be noted. If the body be that of a new-born infant, the degree of maturity should be ascertained (see ii. pp. 190-192).

Internal examination of the body.—If there be any clue as to the cause of death, the cavity supposed to be implicated should be opened first, otherwise the order given below should be followed. In all cases, however, it is important that all the cavities of the body should be opened and their contents examined, since, although a cause of death may be found in the first cavity, yet a counsel defending a prisoner could argue that an earlier cause of death might have been found in one of those left unopened. To open the body the operator should stand on the right side of the cadaver, and should make an incision in the middle line, through the skin and fat, extending from the chin to the symphysis pubis. The abdominal cavity should be opened first, and notes should be made of the relations and appearances of the viscera as seen *in situ*, and of any abnormal conditions of the peritoneum. The position of the diaphragm should then be ascertained on either side by passing the index finger of the right hand to the roof of the diaphragm in the

mid-clavicular line, and then pressing the finger towards the front of the thorax so as to ascertain the height by reference to the ribs.

After opening the thorax, the pericardium should be laid open along its lower and right borders, and notes should be made of the degree of distension of the right auricle and the ventricles, and the state of the serous membrane. Abnormal collections of serum, blood, or other liquid found in any of the cavities of the body should be carefully collected by a syringe or otherwise, and measured in a graduated glass vessel.

Before proceeding further with the examination of the thorax and abdomen, it is desirable to expose the surface of the brain, so that the degree of congestion of its vessels may be ascertained before the great vessels in the thorax or abdomen are cut. To effect this the skull-cap is exposed by making an incision down to the bone from the root of the left mastoid process to the corresponding point on the right side, and then peeling from the skull the frontal and occipital flaps of the scalp. During this proceeding note should be made of any abnormal extravasations of blood that may be found, and careful search should be made for fractures ; if a fracture be found, it should be followed to its whole extent, and any unusual thinness of bone should be noted. To remove the skull-cap the outer table of the skull is sawn through on either side along a line passing anteriorly from a point an inch above the root of the zygoma to just above the frontal eminences, and posteriorly to a point about an inch above the external occipital protuberance. The separation of the skull-cap is carefully completed with the chisel, precautions being taken to avoid fissuring of the bones. The dura mater is then cut through on a level with the edge of the bone, except in the middle line behind, and is turned back so as to expose the surface of the pia mater. In infants under a year old the bones of the skull and the dura mater can be divided together by a strong pair of scissors. The degree of distension of the superficial cerebral veins is to be noted, and the

examination of the abdominal and thoracic cavities should then be proceeded with.

Examination of the abdominal cavity.—The surface of the intestine should first be carefully inspected and palpated, and the condition of the mesenteric glands ascertained. The stomach should then be removed by placing a ligature round the lower end of the œsophagus, and a double one at the beginning of the duodenum, and dividing the œsophagus above its ligature, and the duodenum between the two. The stomach should then be placed on a clean porcelain dish, and opened along the lesser curvature, care being taken that none of the contents are lost. The contents are poured into a clean jar, and the inner coat of the stomach is examined. Note should be made of its colour, and the surface should be examined by the aid of a lens for particles of leaves, berries, colouring matter, crystals, &c., any of which, if found, should be examined under the microscope. Any signs of inflammation, ulceration, corrosion, or perforation should be noted. The œsophagus should then be removed and opened, and any pathological conditions, or signs of corrosive or irritant poisoning, should be noted. The small and large intestines are then to be separately ligatured, removed, and examined in a similar way to the stomach ; note should be made of any pathological conditions, such as ulcers, perforations, growths, and areas of inflammation. The presence or absence of fæces in the lower bowel should be recorded. The liver, spleen, pancreas, kidneys and suprarenal bodies should be removed, weighed, and examined.

The examination of the pelvic organs requires special care in cases of suspected criminal abortion. The peritoneal surface of the uterus, and the upper portion of the posterior surface of the vagina, are carefully examined for perforating wounds, and the vulva is also carefully inspected. The ligaments of the pubic symphysis are then divided with the cartilage knife, and the lower limbs are forcibly separated in order to cause the pubic bones to come apart, and so give room for the separation

of the pelvic organs, which are then removed along with the skin of the perineum and vulva. The bladder is to be opened and examined. The vagina is then cut through with blunt-pointed scissors along the middle of its anterior surface, and its interior and the os uteri carefully inspected. The cervix and body of the uterus are next opened by a continuation of the incision into the vagina, and their interiors carefully examined. The ovaries should be opened by clean cuts, and notes made of any corpora lutea that may be present.

Examination of the thoracic cavity.—The cavities of the heart should be examined before it is removed, and the size of the tricuspid and mitral orifices should be tested by means of the fingers. After removal of the heart, the mitral and aortic valves and the endocardium are thoroughly exposed, and any abnormalities noted. The lungs and their pleural surfaces should be examined previous to removal, and evidence of aeration, collapse, or atelectasis carefully looked for. The lungs, trachea, larynx, floor of the mouth, and tongue should be removed together, and submitted to careful inspection. For the method of examining the lungs in a case of suspected infanticide, see ii. pp. 195-199.

Examination of the cranial cavity.—The brain is removed in the usual way, the spinal cord being cut as low down as possible in order to facilitate the subsequent removal of the remainder of the cord. The brain should then be placed on its ventral aspect, and transverse and vertical sections made through its substance. The sections should not pass quite through the brain, so that the subdivisions may be easily replaced, and held together in order to facilitate the accurate location of any lesion that may be found.

The spinal cord may be removed either from the front by the use of Schmidt's chisels, or from behind. After the surface of the cord has been examined, a series of transverse sections should be made at short intervals from end to end, in order to examine its substance.

The first metatarso-phalangeal and other joints should be

opened to ascertain whether any deposits of sodium urate are present in the cartilages. If necessary, the bones should be examined by freeing them from the soft parts, and then making longitudinal sections with a sharp saw. Evidences of rickets and syphilis should be looked for. When the examination is made several days or longer after death, changes due to decomposition must be allowed for. If the corpse has been some months in the ground or in water, the formation of adipocere will mask the normal appearances, and may be mistaken for tubercular caseation.

Instruments.—The operator should be provided with a pair of scales, a graduated glass measure, some clean glass jars, an ordinary dissecting-case, two pairs of scissors, one pair sharp-pointed and the other blunt-pointed, two full-bellied section knives, a cartilage knife, a saw with a movable back, chisel and mallet, bone forceps, a pair of bowel scissors, a blunt-pointed bistoury, a straight narrow-bladed knife for removing the tongue and floor of the mouth, and a knife with a broad blade about eighteen inches in length for making sections of the brain and other solid viscera. A packing-needle and some twine are required for the final sewing up of the body.

Preservation of viscera for analysis.—For the methods of cleaning the glass jars and afterwards securing them, see p. 113. The jars containing the contents of the stomach and the viscera should be sealed with the private seal of the medical man who has made the necropsy, and to each jar a label should be affixed stating the contents of the jar, the name of the deceased person, the date of death, the date of the necropsy, and the name of the medical man making it. These jars should be delivered personally by the medical man to a responsible party, to whom a list of the jars and contents should be given, a similar list being retained by the medical examiner. If the jars and their contents have to be kept, they must be locked up in a cool place. No preservative should be added to the contents of the jars.

Exhumation.—In the case of the exhumation of a body, it

should be properly identified by the friends or relatives of the deceased before removal from the coffin, at which time, also, the medical inspectors should view it. Exhumations are most frequently made in cases of suspected poisoning. In such cases, the stomach, intestines, liver, spleen, and kidneys should be removed and placed in clean glass jars. If a metallic poison be suspected, the shaft of the femur should be removed. If the body has been buried for long, or if the coffin be decayed, a small portion of the surrounding earth should be taken away for chemical examination.

TOXICOLOGY

CHAPTER VIII

Definition of a poison—Sale of poisons—Noxious things—Conditions affecting the action of poisons—Classification of poisons—Evidences of poisoning—Rules for investigation of a case of poisoning—Preservation of substances for analysis—General treatment of cases of poisoning—Preliminary procedures in the search for poisons.

Toxicology (derived from τοξικὸν, poison, and λόγος, discourse) includes that branch of medical science which treats of poisons, of their history and properties, of their effects upon the living body, their treatment within the body, and their detection.

The accurate definition of the term poison is not an easy matter, and care should be taken not to adopt what is perhaps the popular idea of a poison—viz. that it is a substance capable of injuriously acting on the body when taken or administered *in a small dose,* since many poisons act only as such when taken or administered in large doses. The following is the definition of a poison given by Taylor and Stevenson: 'A poison is a substance which, when absorbed into the blood, is, by its direct action, capable of seriously affecting health or of destroying life.' In connection with this definition, it must be borne in mind that some substances which we regard as poisons do not require to be absorbed into the blood in order to exert their deleterious action; such, for instance, as corrosive acids and alkalies, which act by producing a corrosive effect upon the skin or mucous membrane with which they are brought in contact, and so seriously affecting the nervous system. Wynter Blyth defines poison thus: 'A substance of definite chemical

composition, whether mineral or organic, may be called a poison if it is capable of being taken into any living organism and causes, by its own inherent chemical nature, impairment or destruction of function.' The following is perhaps a fairly clear and comprehensive definition : *A poison is a substance, which, either by its direct action upon the skin or mucous membranes, or after absorption into the blood, is capable of injuriously affecting health or of destroying life.*

It should be borne in mind that the law does not regard the manner in which the substance administered acts, provided a substance is capable of injuriously affecting health or of destroying life. It makes no difference, from a legal point of view, whether the action of that substance on the body is of a chemical or mechanical nature, or whether it produces its fatal result by absorption into the blood or not. For instance, a substance which simply exerts a mechanical action on the stomach or bowels (crushed glass, metal filings, &c.) may, if administered with intent to injure, involve a person in a criminal charge as much as if that person had administered arsenic, or any such poison ; and even supposing that no bodily injury has followed its administration, yet the accused can be tried for 'attempt to commit murder.' The law in relation to this subject (24 & 25 Vict. ch. 100, sec. 11) runs thus : 'Whosoever shall administer, or cause to be administered or taken by any person, any poison *or other destructive thing* with intent to commit murder, shall be guilty of felony.' This is only in accord with common equity, inasmuch as the crime is gauged by the act of administration of the poison, or other destructive thing, and not solely by the consequences that may follow its administration.

It has occasionally been contended or stated by witnesses in trials for attempt to commit murder by administration of poison, that enough poison had not been administered to cause death, or to cause any serious injury, such contention being put forward with the view of endeavouring to lessen the culpability of the person indicted for administering the substance. With

regard to this point, the ruling of Cresswell J. in the case of Hartley (C. C. C., 1850) was to the following effect: 'If poison be administered with intent to murder, it is not necessary there should be enough in the article administered to cause death. If any poison be there, and the intent be proved, the crime of attempting to administer poison is complete.' The Criminal Law Consolidation Act, 1861, has the following provision: 'Whosoever shall, by any other means, other than those specified in any of the preceding sections of this Act, attempt to commit murder, shall be guilty of felony.'

Poison may be administered illegally, not with the intention of committing murder, but with some other criminal intent, such as the employment of drugs in the attempt to procure criminal abortion, or the use of narcotics in attempted rapes. The sections 23, 24, and 25 of 24 & 25 Vict. ch. 100 provide for this:—

'23. Whosoever shall unlawfully and maliciously administer to, or cause to be administered to, or taken by, any other person, any poison, or other destructive or noxious thing, so as thereby to endanger the life of such person, or so as thereby to inflict upon such person any grievous bodily harm, shall be guilty of felony.'

'24. Whosoever shall unlawfully and maliciously administer to, or cause to be administered to, or taken by any other person, any poison, or other destructive or noxious thing, with intent to injure, aggrieve, or annoy such person, shall be guilty of a misdemeanour.'

'25. If, upon the trial of any person charged with the felony above-mentioned, the jury shall not be satisfied that such person is guilty thereof, but shall be satisfied that he is guilty of the misdemeanour above-mentioned, then, and in every such case, the jury may acquit the accused for such felony and find him guilty of such misdemeanour.'

There is also a special provision framed with reference to stupefying poisons, such as chloroform, &c.:—

'Whosoever shall unlawfully apply, or administer to, or

cause to be taken by any person, any chloroform, laudanum, or other stupefying or overpowering drug, matter, or thing, with intent in any such case thereby to enable himself, or any other person, to commit, or with intent, &c., to assist any other person in committing, any indictable offence, shall be guilty of felony.'

The sale of poisons—in small quantities, at all events—is by law restricted to pharmaceutical chemists, chemists and druggists, and medical men ; and in the Pharmacy Act of 1868 poisons are arranged in two groups. With those in Part I. a registration of the sale is compulsory, and the poisons are not allowed by the Act to be sold unless the purchaser is known to, or introduced by some person known to, the seller, the purpose for which the poison is wanted having to be entered in a book provided for the purpose; whereas, those in Part II. merely have to be labelled with the name of the substance, the word 'Poison,' and the name and address of the vendor. The following are the poisons at present arranged in these groups :—

Part I.—Arsenic and its preparations. Aconite and its preparations. All poisonous vegetable alkaloids and their salts. Atropine and its preparations. Cantharides. Corrosive sublimate. Cyanide of potassium, and all metallic cyanides, and their preparations. Emetic tartar. Ergot of rye and its preparations. Prussic acid and its preparations. Savin and its oil. Strychnine and its preparations. Vermin-killers, if preparations of poisons the preparations of which are among the poisons just enumerated.

Part II.—Essential oil of almonds (unless deprived of prussic acid). Belladonna and its preparations. Tincture of cantharides, and all vesicating liquid preparations of cantharides. Chloroform. Chloral hydrate and its preparations. Morphia and its preparations. Corrosive sublimate and its preparations. Nux vomica and its preparations. Opium and its preparations, and preparations of poppies. Oxalic acid. Red precipitate (red oxide of mercury). White precipitate (ammoniated mercury). Vermin-killers containing poisons prepared for the destruction of vermin, if not subject to the provisions of Part I.

Noxious thing.—With regard to the definition of a noxious thing, the law throws the responsibility on a medical witness of expressing an opinion as to whether the substance given was a noxious thing, and as to whether it was given in excess, and so was likely to produce annoyance or injury to health. In *Reg.* v. *Hennah* (Cornwall Ass., 1877), it was contended in defence that, if what was administered could produce no effect, it would not in law amount to administering a noxious thing as required by the statute. On that occasion Lord Chief Justice Cockburn ruled that, unless the thing was noxious in the quantity administered, it cannot be said that there has been a noxious thing administered; and he further said that there must be a distinction between a thing only noxious when given in excess, and a thing which is a recognised poison, and is known to be a thing noxious and pernicious in its effect. While this ruling remains, medical witnesses must be careful in considering and expressing an opinion as to the quantity of the substance administered, and whether such quantity was likely to injure or annoy a person.

Conditions affecting the Action of Poisons

As a medical witness may be called upon to state the dose of the substance required to be poisonous or noxious, it is important that the various conditions affecting the toxic action of poisons should be carefully considered. These conditions are:—(1) Habit; (2) Idiosyncrasy; (3) Age; (4) State of health; (5) The condition in which the poison is administered or taken; (6) The mode of introduction into the system, and the amount of food present in the stomach at the time.

1. **Habit.**—The influence of habit on many poisons may very considerably diminish their effects; for instance, the constant taking of opium causes considerable loss of its toxic and therapeutical powers, and necessitates the employment of a much larger dose to bring about its physiological action. Opium eaters and drinkers, and persons addicted to the use of morphine, are able to take in one dose a quantity of the drug

which would assuredly prove fatal to anyone not so addicted to its use. This tolerance on the part of the system to the action of poisons, as a result of their acquired and prolonged use in small doses, is not peculiar to opium or morphine; arsenic, alcohol, chloral hydrate, strychnine, and cocaine are examples of poisons in connection with which such tolerance may be acquired. The Styrian practice of arsenic-eating is a fairly well known example of this. Cases are published of Styrians eating five grains of arsenic in one dose, whereas a considerably smaller dose would probably prove fatal to an adult not addicted to this practice. It is, however, probable that amongst those Styrians who have adopted this practice of arsenic-eating, symptoms of acute arsenical poisoning sooner or later show themselves.

2. **Idiosyncrasy.**—Idiosyncrasy is different from habit, in that it does not diminish the effect of a poison. Some persons are so constitutionally disposed as to be intolerant of small doses of poisons or other substances, which in the same quantities would not injuriously affect the majority of beings. Some persons are naturally tolerant of large doses of poison, and others intolerant of small doses of the same poison. For instance, in some people an ordinary medicinal dose of arsenic or of mercury may exert a toxic action; certain articles of food may exert peculiar effects upon certain individuals: such, for instance, is the case with some kinds of shell-fish and pork, which may not contain anything poisonous, and which the majority of people may be able to eat without the production of any ill effects. From the medical point of view the subject of idiosyncrasy is important, since, after the consumption of a particular kind of food, symptoms resembling those of poisoning may occur.

3. **Age.**—In the majority of cases children are more easily affected by poison than adults, and such is especially the case with opium and the preparations of morphine. On the other hand, certain poisons, like belladonna, are better tolerated by children than by adults.

4. **State of health.**—A disordered or diseased condition of the body may in some cases render a person less susceptible to the action of certain poisons; whereas in other cases it may increase their toxic effect and cause small doses to exercise a fatal action. Patients suffering from dysentery, tetanus, acute mania, and delirium tremens can take large doses of opium or morphine which might be sufficient to kill an adult in ordinary health; on the other hand, patients suffering from the different forms of Bright's disease and apoplexy are much more susceptible to the effect of opium or of the preparations of morphine; so that a dose of opium or morphine, which can be borne by an adult in ordinary health with impunity, may kill a person suffering from one of those diseases. If a person be suffering from gastro-intestinal catarrh, small doses of any irritant poison would aggravate the already existing condition. It should be carefully borne in mind that the substitution of poisons for medicines, or the addition of poisons to medicines, with a criminal intent, is not unusual, and a medical practitioner is especially apt to be misled when poison is secretly administered in medicine to a patient already suffering from symptoms of gastro-intestinal catarrh. The points that are especially indicative of suspicion in such cases are the increase in the gravity of the symptoms without there being anything in the condition of the patient to explain such an increase, or the supervention of fresh symptoms which might indicate the nature of the substance being administered, especially when such aggravation or occurrence of fresh symptoms occurs after the taking of medicine or of food. In the event of such suspicion being raised in the mind of the medical attendant, his duty is to employ trained nurses who should be in attendance both day and night on the patient. In some cases only a chemical examination of the suspected articles may actually reveal the fact of the criminal act of the administration of poison.

5. **The condition in which the poison is administered.**—The condition of a poison especially with regard to its solubility,

very considerably affects the rapidity with which it produces its toxic action. If a poison be in solution, it is more rapidly absorbed into the circulation and more quickly produces its toxic action than if administered in a solid or compact form; for instance, if strychnine be administered in hard pills, its poisonous action may be considerably delayed, whereas if administered in a state of solution the toxic effect is produced in a much shorter time.

6. **The mode of introduction into the system.**—Poisons may be introduced into the system in various ways. The commonest way is by the mouth, the absorption of the poison afterwards taking place from the stomach and intestines; this is a fairly rapid way of producing the toxic effects of a poison, but the amount of food that may be present at the time in the stomach may very considerably affect both the rate of absorption into the system and the action of the poison. A poison taken upon an empty stomach is more rapidly absorbed, and produces a quicker and more powerful toxic action, than if taken on a full stomach.

Another method by which poisons may be introduced into the system is by hypodermic injection, by which method, on account of the rapid introduction of the poison into the circulatory system, the toxic action is, as a rule, speedily produced. The rapidity of absorption is very great; after the hypodermic injection of a solution of a poison, it becomes diffused throughout the whole circulation in a few seconds.

Again, poisons may be absorbed from the unbroken skin, though such absorption is necessarily very slow, or from ulcerated surfaces upon the skin, when the absorption is somewhat more rapid, or from the various mucous membranes, such as by injection into the rectum or vagina, and by application to the nasal mucous membrane, or to the mucous membrane of the throat; in addition, a poison may be introduced in the form of vapour into the lungs, and so gain access to the system, as in the case of ether or chloroform administered by inhalation.

Poisonous Doses

The medical witness is frequently asked what dose of a poison would be sufficient to cause death. In answering such a question, not only should the average toxic dose be given, but also the smallest dose that has been known to produce death in an adult or a child, as the case may be, since, if such a dose has once produced a fatal result, it may produce it again. It should be carefully borne in mind that, in cases of death from poisoning, in which poison has been extracted from the contents of the stomach, the portion of poison remaining in the stomach, and extracted by the analyst, is not the portion which produced death, but merely the residue or surplus of the poison which has undergone absorption into the circulation, the latter portion being mainly responsible for the fatal result; so that the quantity which may be found in the stomach or intestines, or deposited in the different viscera and tissues of the body, gives no true idea of the quantity that has actually been taken by, or administered to, the deceased, especially as a considerable amount may have been removed by vomiting, purging, and in the urine. Moreover, death may result from the effects of the poison, and yet no trace of the poison may be found in the stomach, intestines, or any of the viscera. For instance, death has been known to result from strychnine poisoning with all its characteristic symptoms, and yet sufficient time has occurred for the elimination, and perhaps the partial destruction, of the poison, previous to the occurrence of death, so that the question as to whether the amount of poison discovered in the body is sufficient to have caused death is in reality a matter which should not affect the case. As Orfila has remarked with regard to arsenic, 'That portion which is found in the stomach is not that which has caused death; but the surplus of the quantity which has already produced its fatal effects by its absorption into the system.' If, however, a very small quantity of a poison be found in the body, the question may fairly be raised whether

that small amount is not the remains of a medicinal dose of the poison legitimately administered or taken.

Classification of Poisons

The classification of poisons is not a matter of much importance or of much utility. A common method of classifying them is in the following groups :—

1. **Inorganic poisons.**
   I. Irritant poisons.
   II. Corrosive poisons.
2. **Organic poisons.**
   I. Irritant poisons.
   II. Poisons affecting—*a.* the Heart ; *b.* the Lungs ; *c.* the Brain ; *d.* the Spinal cord.

Perhaps a more useful classification is the arrangement of poisons according to the way in which they produce death ;

Irritant poisons	Poisons arresting respiration	Poisons arresting the action of the heart
Sulphuric acid	Calabar bean	Antimony salts
Hydrochloric acid	Cantharides	Alcohol
Nitric acid	Colchicum	Chloroform
Caustic potash	Conium	Chloral
,, soda	Lobelia	Belladonna
Ammonia	Ergot	Stramonium
Lime	Aconite	Henbane
Copper salts	Strychnine	Digitalis
Mercury salts	Hydrocyanic acid	Veratria
Lead salts	Opium	Tobacco
Silver salts	Tobacco	Barium salts
Zinc salts	Chloroform	
Arsenic and its preparations	Nitrous oxide	
Tin salts		
Chromium compounds		
Phosphorus		
Carbolic acid		
Oxalic acid		
Savin		
Cantharides		

but in connection with this classification it should be borne in mind that the same poison frequently affects more organs than one, and therefore in connection with the list given on p. 103, it should be remembered that, though the organ referred to in the column containing any one poison is the one mainly responsible for the cause of death, yet it may not be the only one affected by the poison in question.

Corrosive poisons.—These poisons destroy, by direct chemical action, the tissues with which they come in contact. Many irritant poisons are corrosives also; all corrosive poisons are necessarily irritants. Corrosive poisons produce an effect in the act of swallowing, there being felt immediately an acrid or burning taste extending from the mouth down the gullet to the stomach; speedy vomiting is also induced in the majority of cases, and the vomited matters may be peculiarly stained or affected by the nature and action of the poison, in the case of corrosive acids and alkalies; the symptoms come on immediately, as mere contact of the poison with the mucous membranes is sufficient to produce destruction of the latter on account of the ready destructibility of tissues by corrosive acids and alkalies. It is important to carefully examine the mouth and throat in cases of poisoning or of suspected poisoning of this kind.

Irritant poisons.—These poisons, by their direct irritation of the mucous membranes with which they come in contact, set up inflammation; as time is required for the production of this inflammation, the symptoms do not as a rule come on until half an hour to an hour, or longer, after taking the poison. The first symptom produced, as a rule, by an irritant poison is pain in the stomach and bowels, due to the inflammation set up, which pain is generally referred at first to the epigastrium; vomiting and purging as a rule soon occur, and, later on, as the poison becomes absorbed into the system, symptoms indicative of its action on the nervous centres, or on the heart, may appear.

Acute poisoning.—If the destruction of life by poisoning be rapid, it is called acute poisoning.

Chronic poisoning.—If injury or death occur from the slow action of poison, it is called chronic poisoning. Most poisons, which in large doses produce acute poisoning, may, if given in small and repeated doses, gradually kill or undermine the health of the patient and so may act as chronic poisons.

Evidences of Poisoning in the Living Subject

The detection of poisoning in the living subject is a matter of great importance to the medical man, as otherwise the symptoms produced by a poison may be mistaken for those resulting from disease, and, in consequence of the non-employment of the antidotes and treatment necessary to counteract the effects of the poison, the death of the patient may result. Moreover, in cases in which a poison proves fatal, the symptoms produced by it may form an important part of the evidence as to its nature or identity, although it should be remembered that there are no absolutely characteristic symptoms of any poison. The following are the recognised and classical rules by which the symptoms of poisoning may generally be distinguished from those of disease.

1. **In cases of poisoning the symptoms as a rule are sudden in their onset, and generally occur while the patient is in health.**

2. **In cases of poisoning the symptoms frequently appear either after a meal, or after the taking of some food or medicine. This constitutes, perhaps, the most important indication of poisoning in the living subject.**

3. **If several people eat at the same time of the same food or article, mixed with poison, they are all seized with similar symptoms.**

4. **The recognition, perhaps, of a case of poisoning may only be made sure of by the detection of the poison in some of the food that has been partaken of, or in the vomited matters.**

These rules will now be briefly discussed.

I. With regard to the sudden appearance of symptoms in cases of poisoning, it should be borne in mind that they do not necessarily begin immediately after taking the poison. In cases of poisoning by hydrocyanic acid, oxalic acid, strychnine and corrosives, the symptoms generally appear immediately, or within a few minutes after the swallowing of the poison; but with poisons generally, the symptoms are not manifested till half an hour to an hour or longer has elapsed. Moreover the condition of the body with regard to disease may very considerably affect the incidence of the symptoms (see paragraph 'State of Health,' p. 100).

II. In connection with the appearance of the symptoms soon after a meal, or after something has been partaken of, it should be remembered that perforation of the walls of the stomach by a gastric ulcer is most likely to occur after the ingestion of food, and that the symptoms produced by the perforation and the escape of food into the peritoneal cavity are likely to very closely simulate those of irritant poisoning. Moreover the poison may be introduced into the system in some other way than by the stomach (see p. 101). The diseases which most simulate irritant poisoning are cholera (simulating poisoning by arsenic or antimony), ulceration of the stomach, gastritis, gastro-enteritis, peritonitis, and intestinal obstruction. Those which most resemble neurotic poisoning are cerebral hemorrhage (which may simulate opium poisoning), epilepsy (which may resemble hydrocyanic acid poisoning), and tetanus (simulating strychnine poisoning).

III. As to the seizing of several persons with similar symptoms after partaking of the same food, it must be carefully borne in mind that symptoms very similar to those produced by metallic irritant poisons may be produced by food which has been rendered toxic by putrefactive or fermentative changes. This specially occurs in connection with mussels, pork, sausages, and some other kinds of food. In such cases the poisonous effects are generally due either to a bacillary infection of the article of food, or to the production within it of a toxic substance

which is usually either a ptomaine or albumose (see chapter on Poisonous Foods, p. 407).

Evidence of Poisoning in the Dead Body

The additional evidence as to poisoning that is obtained after the death of a person may be best discussed under the three following heads :—

1. The period after the first occurrence of symptoms at which death has taken place.

2. The evidence that may be obtained from a post-mortem examination of the body.

3. The evidence that may be obtained from an analysis of the contents of the stomach, and of the various viscera.

I. The period at which death takes place after the first occurrence of symptoms is a matter of great importance, as fatal doses of the commoner poisons usually produce death within fairly definite periods of time. The medical witness is frequently asked to state the usual period of time within which a poison proves fatal ; this period may of course be very much affected by the quantity of poison taken, and the form in which it was administered (whether in solution or not). The average period of time that a poison takes to kill may certainly be given by the medical witness, but it is advisable to lay more stress upon the shortest time in which a certain poison has produced death, since, if it has once produced death in such a time, it may do so again.

II. A post-mortem examination of the body is one of the most important means of deciding, or of suspecting, whether a person has died from poison. The external examination of the body is not of much use in furnishing a clue as to whether it is a case of poisoning or not. The old idea that the bodies of persons who were poisoned putrefied more rapidly than those who have died from natural causes is an entirely erroneous one, but in connection with cases of poisoning it must be borne in mind that, excepting the corrosive poisons, which always leave

evidence of their introduction into the system, other poisons may destroy life without leaving any changes that can be detected by the naked eye at the post-mortem examination; such cases, however, are not very common, but in the event of such the proof of poisoning has to rest upon the analytical examination.

It is, of course, a matter of great importance that the appearances and changes produced within the body as a result of disease should not be confounded with those produced by means of poison. These appearances and changes are mainly to be met with in the stomach and intestines, and consist of—(*a*) Redness; (*b*) Ulceration; (*c*) Softening; (*d*) Perforation. As each of these conditions may be produced either by disease or by poison, it will be well to discuss the methods by which they may be distinguished according to their causation.

(*a*) **Redness.**—Redness of the mucous membrane of the stomach and intestines is usually produced by irritant poisons, as a result of the inflammation set up by them; the redness is generally of a deep crimson colour, which becomes brighter when the mucous membrane is exposed to the air; the colour may be diffused over the whole mucous surface, or it may be in irregular patches, dots, or striæ, but it is usually most marked about the cardiac end of the stomach. The redness of the mucous membrane of the stomach and intestines may be due to inflammation as a result of disease, as in the various forms of gastritis and gastro-enteritis. In many cases only a knowledge of the symptoms which preceded death, or the chemical detection of the poison in the contents of the stomach or in some of the other viscera, would enable a medical man to decide whether death was due to natural disease or to poisoning. In the healthy state the mucous membrane of the stomach is pale and grey, except during digestion, when it is slightly reddened, and this slight redness may remain in the stomachs of those who have died somewhat suddenly whilst digestion was going on. Moreover, as demonstrating the fact that colour alone should not be taken as an indication of inflamma-

tion, it is to be remembered that, where the stomach has been in contact with the liver or the spleen after death, it frequently acquires a deep reddish-brown colour from the transudation of blood, and a similar colour may be produced by the gravitation of blood to the posterior walls of the stomach or intestines after death. In addition, some articles of food or of medicine containing colouring matter may have been taken ; some fruits, for instance, colour the gastric mucous membrane, and copious draughts of iced water are also stated to have produced reddening of the membrane of the stomach.

(*b*) **Ulceration.**—In cases of corrosive poisoning, ulceration or corrosion of the mucous membrane of the stomach is common, and ulceration is also found on rare occasions in connection with irritant poisoning ; but ulceration of the stomach is a far more common result of disease than of the action of poison. The distinguishing features of an ulcer of the stomach produced by disease, and an ulcer the result of poison, are as follows :—

Ulceration produced by disease.—In such a case the mucous membrane is only inflamed in the neighbourhood of the ulcer. The ulcer, as a rule, is situated on or near the lesser curvature of the stomach ; it generally presents a clean punched-out appearance, and, as a rule, is of moderate size. The edges are raised and hardened, being frequently more or less cartilaginous to the touch. The ulcer may extend only through the mucous membrane to the muscular walls, or it may pass through the muscular coats to the peritoneum, or it may perforate the peritoneum, and so produce a perforating gastric ulcer. Its shape is generally conical, the widest part of the cone being situated next to the mucous membrane, and the narrow part of the cone being towards the outer wall of the stomach.

Ulceration produced by poison.—The redness of the mucous membrane is diffused, and is not limited to the immediate neighbourhood of the ulcer. The edges of the ulcer are not, as a rule, hardened and raised, but are usually soft and ragged ; the ulcer is not conical in shape, but its outline is generally irregular.

(*c*) **Softening.**—This, as a result of poisoning, is generally produced by corrosive poisons, so that in cases of softening resulting from poisons, the effects of the corrosive action will generally be found in the mouth, throat and gullet. Of the corrosive poisons, the alkalies are especially apt to produce softening.

(*d*) **Perforation.**—The stomach may become perforated from the following causes :—(1) The action of a corrosive poison ; (2) Disease ; (3) The post-mortem solvent action of the gastric juice. The following are the distinguishing features of these different modes of perforation of the stomach :—

1. *Perforation produced by poisons.*—This especially occurs in connection with poisoning by the strong mineral acids, and of these sulphuric acid is the commonest cause of perforation of the stomach. In connection with perforation caused by sulphuric acid, the walls of the stomach are blackened and usually extensively destroyed.

2. *Perforation caused by disease.*—This is by no means an uncommon condition ; the diagnosis of it is important, since the effects of perforation of the stomach by disease are very apt to be mistaken for acute poisoning, as a gastric ulcer is most liable to perforate suddenly and soon after taking a meal. The symptoms may come on while a person is in the apparent enjoyment of good health, and the sudden severe abdominal pain and collapse, which are the result of the escape of the contents of the stomach into the peritoneal cavity, may closely simulate the symptoms of acute poisoning. If the perforation be the result of a gastric ulcer produced by disease, it is readily recognised at the post-mortem examination ; the aperture is oval or rounded and of small size, the situation is near the lesser curvature of the stomach, the edges are smooth and thickened, and the ulcer is funnel-shaped, the wide part of the funnel looking into the interior of the stomach, and the narrow part being situated at the base.

3. *Perforation due to the post-mortem solvent action of the gastric juice.*—In perforation of this nature the aperture is

situated on the posterior wall of the stomach (provided the corpse has been lying on the back); it is very large, of an irregular form, and has ragged pulpy edges; the mucous membrane is not inflamed, and the edges of the perforation are free from the colour-changes produced by the action of corrosive poisons.

Rules for the Investigation of a Case of Poisoning

When called to a case of poisoning, if the patient be alive, the first duty of the medical man is to use every effort to save the life of the individual. Full directions as to the treatment that should be employed, and the antidotes that should be administered, will be given in connection with the individual poisons. But, in addition, it is the duty of the medical man to note down any circumstances which may lead to the detection of the perpetrator of the crime. The following are the points to which the attention of the medical attendant should be chiefly directed, apart from the treatment of the patient:—

1. The time at which the symptoms commenced, and the nature of the symptoms.

2. The time at which the symptoms commenced after the last ingestion of food or medicine.

3. The occurrence of any recent previous illness from which the patient may have suffered.

4. If the patient has vomited, the vomit should be collected, or, if necessary, scraped up from the floor or from the dress, bedding, or carpet; if necessary, a portion of the dress, bedding, or carpet containing the vomit should be cut out and preserved.

5. The nature of the food recently taken by the patient should be ascertained, and if suspicion attaches to any articles of food, these should be secured by the medical man and preserved under seal.

If the death of the patient occur, in addition to attending to the points just mentioned, note should also be made of the following:—

(1) The exact time at which death occurred.

(2) The position of the body with regard to surrounding objects; its attitude, and the condition of the dress.

(3) All surrounding objects should be carefully observed, and any bottles, packets, or weapons in the room should be collected and preserved.

(4) The condition of the body as to lividity or pallor should be noted, and also whether the countenance presents a distressed or calm appearance.

In connection with the post-mortem examination of a suspected case of poisoning, especial attention should be directed to the following points:—(i) A note should be made of the time after death at which the post-mortem examination is being made. (ii) The presence or absence of rigor mortis should be noted, and if present, the parts affected by it. (iii) The external appearance of the body, whether livid or pallid, should be noted, and whether marks of violence or of blood are present. (iv) The state of the abdominal viscera should be carefully observed; if the stomach and intestines are opened, the exact seat of any inflammation that may be found should be carefully noted, and the contents should be preserved in separate vessels. (v) If the stomach be left unopened, it should be ligatured at both ends, removed, and placed in a clean glass jar; if it be opened, the contents should be poured into a graduated vessel, and a note made at the time of the quantity, odour, colour, reaction to test-paper, and anything peculiar or noticeable as to the nature of the food. (vi) The liver and gall-bladder should be removed and preserved in clean vessels for chemical examination. (vii) The bladder, with any urine in it, should be removed, and the urine preserved in a clean bottle for the purposes of analysis. (viii) All the other organs of the body, including the heart, trachea, lungs, œsophagus, and brain, should be carefully examined.

It is most important that attention should be given to the last-mentioned point, since one great object of the examination of any case of death from poisoning is to show that the person could not have died from any natural cause, and in connection with cases of poisoning, counsel defending accused persons

generally give a rigorous cross-examination as to the condition of the heart, lungs, and brain, and if any of those organs have not been properly examined, they may contend that the cause of death may have been present in the organ not subjected to examination. For the detailed method of conducting the post-mortem examination, see p. 87.

Identity of substances preserved for analysis after a post-mortem examination in a case of suspected poisoning.—It is the duty of the medical man to carefully secure and seal vomited matters, as well as the liquids and viscera taken from the body of the deceased, in clean vessels, and to affix a label to each vessel stating the nature of its contents, the name of the deceased, and the date of removal; the signature of the medical man should be added to every label. This is necessary, as the law insists that proof of the identity of the various articles should be forthcoming. The vessels in which the viscera are secured should be wide-mouthed glass jars, which should be thoroughly cleaned out by being first scoured with sand, then rinsed with strong hydrochloric acid, and afterwards thoroughly washed with water. No antiseptic should be added, and the vessels should be covered over with oil-silk, which should be tied down, and a seal affixed to the string. Calico, linen and paper of all sorts should not be employed for covering the vessels, since these articles may have arsenic or lead in the dressings that have been used in their preparation.

General Treatment of Cases of Poisoning

In the treatment of a case of poisoning, the following are the various modes of procedure :—(i) Either remove the poison from the stomach, or neutralise it, or render it insoluble by the administration of suitable antidotes. (ii) Overcome the effects of that portion of the poison which has been absorbed, and promote its elimination by suitable treatment. (iii) Alleviate any dangerous symptoms, and endeavour to keep the patient alive till the effects of the poison have passed off.

These methods of treatment require brief discussion.

I. The removal of the poison from the stomach may be effected either by the induction of vomiting by emetics or otherwise, or by employing the stomach-pump or stomach-tube. Vomiting may be induced by emetics, of which perhaps the safest is a dessert-spoonful or tablespoonful, according to the age of the patient, of flour of mustard stirred up in about half a tumblerful of water, and given to the patient to drink; other emetics are sulphate of zinc in doses of fifteen to thirty grains, the hypodermic injection of one-tenth of a grain of apomorphine, powdered ipecacuanha in fifteen to thirty grain doses, carbonate of ammonia in twenty to thirty grain doses, sulphate of copper in five to ten grain doses, and copious draughts of tepid water.

Poison may also be removed from the stomach by means of the stomach-pump, provided that instrument be handy, and this has the advantage of enabling the operator at the same time to thoroughly wash out the stomach of the patient. When employing the stomach-pump, the patient should if possible be made to sit in a chair with the body partially reclining; if this be impossible, the person may be laid in the recumbent position, and the gag provided with the stomach-pump should then be placed between the teeth, and held in position by an assistant standing behind the patient. The end of the tube of the pump should be smeared over with vaseline or glycerine, and passed through the central hole of the gag down the œsophagus into the stomach; this can be easily effected if the end of the tube be first bent into a slightly curved position, and then passed well to the back of the pharynx and kept in contact with its posterior wall while it is being pushed down. When the end of the tube has been passed into the stomach, the pump should be attached, and before any of the contents of the stomach are removed, a pint of warm water should be injected. The pump is then worked so as to withdraw about a pint of the contents from the stomach, and then a second pint of warm water is injected and a similar amount withdrawn; this is repeated

four or five times. In this way the stomach is prevented from being at any time entirely empty, as otherwise injury to its coats might result.

In the place of the stomach-pump, the removal of the contents and the washing out of the stomach may be effected by syphonage by means of the stomach-tube. For this purpose about five feet of india-rubber tubing of the size of the little finger are required, with a glass funnel attached to one end. The free end of the tube is passed down the œsophagus into the stomach, the passage being readily effected as soon as the tube gets into the grip of the constrictors of the pharynx. The funnel is then held as high as the tube will allow, and a pint of warm water is gradually poured down it into the stomach; before the last portion of water has left the funnel the tube just below it is tightly pinched between the finger and thumb of the operator, and the funnel is lowered below the level of the patient's stomach and held over a basin or pail; the contents of the stomach then flow over by syphon action. The funnel is again raised, and the process of pouring in warm water and removing it by syphonage is repeated four or five times. In cases of poisoning with corrosives, it should be carefully borne in mind that neither emetics nor the stomach-pump should be employed, as rupture or perforation of the walls of the œsophagus or of the stomach is liable to be the result.

Antidotes may be administered in order to neutralise the chemical action of the poison, or to render it insoluble and inert, or to produce a physiological antagonism to its action, or simply to exert a mechanical action. The various antidotes will be described in connection with the different poisons.

II. The effects of that portion of the poison which has been absorbed may be counteracted by suitable treatment. With those poisons which tend to produce asphyxia, artificial respiration may be resorted to; with those that tend to depress or stop the action of the heart, cardiac stimulants can be used; in cases of narcotic poisoning the patient may be roused by being kept moving; and in the cases of those poisons which produce

convulsions by excessive stimulation of the nervous centres, sedatives can be employed. The means employed to overcome the effects of poisons after absorption, and to promote their elimination, will be fully described in connection with the various poisons.

III. The modes of alleviating dangerous symptoms in cases of poisoning, and of keeping the patient alive till the effects have passed away, will also be fully described in connection with the individual poisons.

Method of Procedure in the Examination of the contents of the Stomach, Viscera, &c., for Poison

Notes should be made of the number of jars or vessels received, the condition in which they are received, whether sealed, and whether the wrappers are likely to have contaminated the contents. Copies should be made of the descriptions of the contents of the jars as given on any affixed labels. After opening the jars and vessels, notes should be made of the appearance, smell, colour, and reaction to test-papers of the contents; the weight of solid substances and the volume of liquids should be determined. The jars, seals, wrappers and labels should be preserved for production, if required, in a court of law.

If the stomach be unopened and tied at the œsophageal and pyloric extremities, it should be cut open and the contents transferred to a shallow evaporating dish. Careful examination should be made as to the nature of the food, the colour of the contents, and the presence of any peculiar smell; and a minute search should be instituted for any abnormal constituents, such as white or coloured powders, seeds, leaves, &c. The stomach contents should then be transferred to a graduated glass vessel, and their volume determined. The stomach itself should next be laid on a clean dish or sheet of glass, and the mucous coat carefully examined first with the naked eye and then with a

lens, note being made of any pathological appearances, such as congestion, inflammation, thickening, softening, erosion, ulceration, perforation, or hæmorrhages; careful search should also be made for the presence on the mucous membrane of particles of powders, crystalline fragments, seeds, leaves, &c. The mucous surface of the stomach should next be washed with distilled water, the washings being added to the contents of the stomach.

In the majority of cases where a toxicological investigation is required, there is some clue as to the nature of the poison, and direct search is then made for that poison; the presence or absence of other poisons, however, should always be determined. If there be no clue as to the nature of the poison, a systematic analysis must be undertaken. The duty of the toxicologist is not only to discover the poison in the contents of the stomach of the deceased (since it may have been introduced there after death), but also to detect it, in the absorbed state, in the viscera. Exhaustive details as to the analytical procedures would be out of place in this work, but full accounts of the various tests will be found in connection with the descriptions of the different poisons, and the following is an indication of the method of procedure when a systematic analysis is required.

I. Volatile poisons, such as hydrocyanic acid, alcohol, chloroform, &c., may be separated by distillation of the contents of the stomach, or of the finely divided viscera, after acidulation with tartaric acid.

II. Arsenic is best separated from the viscera, &c. by drying the organic substance and distilling with strong hydrochloric acid (see p. 168).

III. Other metallic poisons may be tested for, either before or after the destruction of organic matter. If the organic matter is to be destroyed, the following process, known as the *moist method*, is the preferable one:—The contents of the stomach, or the viscus reduced to a pulp, are mixed with water to the consistence of thin gruel, and then placed in a large flask with some crystals of potassium chlorate. About

half an ounce of the chlorate is required to each pound weight of the liquid mixture. Pure hydrochloric acid is then added, and the flask gently heated on the water-bath, when a mixture of chlorine and the oxides of chlorine is evolved, which breaks up the organic matter, and converts any mineral poison that may be present into a chloride. If necessary, either more hydrochloric acid or more potassium chlorate should be added, until the liquid becomes limpid, or assumes a light yellow colour. It is then transferred to an evaporating basin, and heated on the water-bath until the smell of chlorine has entirely disappeared, when it is filtered hot. After the filtered liquid has cooled, a stream of sulphur dioxide is passed through it to reduce any metals in the higher state of oxidation to the lower state. If the liquid be filtered hot, as advised, lead, if present, will pass into the filtrate, as chloride of lead is very appreciably soluble in hot water. The only metallic poison that would escape detection by this process is silver, the chloride of which is insoluble in both cold and boiling water, and so would not pass through into the filtrate. It is therefore necessary to make a special examination for silver.

IV. Alkaloidal poisons must be separated and purified by the general process for the extraction of alkaloids (see p. 254).

The detection of absorbed poison in the viscera is in the great majority of cases positive evidence of absorption during life, but it should be remembered that post-mortem imbibition can occur. Thus, if a solution of a poison be introduced by means of a tube into the stomach of a corpse, the liquid can penetrate the walls of the stomach by osmosis, and thence pass into the adjacent viscera. Poison may also be introduced into the body after death in the process of embalming, such as by the injection of arsenical or mercurial solutions into the blood-vessels.

Poisons

The different poisons will now be fully considered and described, employing in each case the following order :—

1. The symptoms produced by the poison.
2. The treatment and antidotes to be employed.
3. The post-mortem appearances resulting from a lethal dose of the poison.
4. The fatal dose of the poison.
5. The fatal period or time that elapses before death occurs.
6. The tests by which the poison may be detected.

INORGANIC POISONS

CORROSIVE POISONS

CHAPTER IX

Poisoning by sulphuric acid—Nitric acid and its fumes—Hydrochloric acid—Oxalic acid and salt of sorrel—Caustic potash—Caustic soda—Ammonia.

Sulphuric Acid

Sulphuric acid, or oil of vitriol as it is commonly known, is more extensively employed in the arts and manufactures than any other acid. It is occasionally taken for the purpose of suicide, especially by women. Poisoning with sulphuric acid is perhaps more commonly the result of accident, as the acid is frequently kept in ginger-beer and other bottles, and so is accidentally swallowed by children. Apart from poisoning by sulphuric acid, injury to the person may be the result of vitriol-throwing, which is occasionally resorted to by persons in fits of jealousy or of rage. Vitriol-throwing is more common on the Continent than in this country, and is punished by law as a felony.

Symptoms.—If sulphuric acid be swallowed in a concentrated form, the symptoms come on immediately. They are:—(i) Violent burning pain in the throat and œsophagus down to the stomach; the pain is very severe and the body is generally bent to relieve the tension of the corroded œsophagus and stomach. (ii) Retching and vomiting speedily occur, the vomited matters are generally frothy, and contain a dark coffee-groundlike substance mixed with blood, and shreds of dis-

coloured mucous membrane. (iii) The mouth is excoriated from the corrosive action of the poison; the surface of the tongue is at first whitened, and later on assumes a brown colour. In those rare cases in which sulphuric acid has been given in a spoon to an infant, the mouth may escape excoriation, from the acid having been passed at once to the back of the fauces. (iv) Great difficulty of breathing occurs; the respiration is noisy and the face is generally cyanosed. (v) Great thirst is experienced; swallowing is difficult or impossible on account of the corrosion and swelling of the membrane of the pharynx and œsophagus, and the consequent pain in the attempt to swallow. (vi) Obstinate constipation occurs in almost all cases, but on rare occasions diarrhœa has been produced.

The dress of the patient should be carefully examined for spots of staining or corrosion with the acid.

Treatment and antidotes.—The employment of emetics and the use of the stomach-pump should always be avoided. The first object is to neutralise the acid that has been swallowed; for this purpose the administration of half an ounce to an ounce of magnesia (magnesium oxide) in a tumblerful of water forms the best antidote. If magnesia be not at hand, some form of calcium carbonate or chalk should be used; this is to be obtained in most houses in the form of *whiting*, which is used for polishing plate; half an ounce to an ounce of it should be crushed up and given in a tumblerful of water. If whiting be not procurable, the plaster removed from a ceiling or cornice may be crushed up and administered in water; carbonate of soda (washing soda) may also be employed to neutralise the acid. If none of these substances can be procured, plenty of water may be given to drink in order to dilute the acid, and so lessen its corrosive and irritant action.

A hypodermic injection of morphine should be administered to alleviate the pain. The further treatment consists in the employment of demulcent and mucilaginous drinks, such as linseed tea, milk and barley water, milk and lime water, almond mixture, or olive oil emulsion. The employment of nutrient

enemata may be required in cases in which the patient is unable to take food by the mouth. The burns on the skin should be treated by the application of linseed oil and lime water liniment. In rare cases where dyspnœa is urgent and death is threatened by suffocation, the operation of tracheotomy may be necessary.

Post-mortem appearances.—If the case has been a rapidly fatal one, the mucous membrane of the throat will be found white and softened. The mucous membrane of the œsophagus is generally corroded and of a brownish-black colour; cases, however, have been recorded in which the œsophagus has escaped the action of the poison, although the latter has passed down it into the stomach. The stomach is found collapsed, constricted, and frequently perforated; the contents are of a tarry nature and consist in great part of mucus and blood, the latter being partly changed into hæmatin. The mucous membrane of the stomach may be slimy in consistence, or corrugated, or removed in parts; it is dark-coloured or even black in patches, the parts surrounding the corroded patches showing signs of inflammation. On removing the blackened membrane from a patch, the red colour of inflammation may be seen in the part beneath. Signs of the corrosive action of the poison may be found in the duodenum. In the blood-vessels clots have been found. In some cases of sulphuric acid poisoning pathological changes have been observed in the kidneys, consisting of destruction of the renal epithelium.

Sulphuric acid may act fatally, without reaching the stomach, by passing into the larynx and causing so much swelling of the parts as to close the air-passages, and so produce death by suffocation; this has been observed in children.

Fatal dose.—The fatal effects of sulphuric acid are due rather to the strength of the acid than to the quantity taken; the amount of food that may be present in the stomach at the time also materially affects the action of the acid, so that a dose of sulphuric acid which might prove fatal if taken on an empty stomach, would probably not have a similar effect if taken upon

a full stomach. The smallest dose of sulphuric acid that has proved fatal occurred in the case of a child, one year of age, and consisted of half a teaspoonful or about forty minims. The smallest quantity that has proved fatal to an adult is one teaspoonful or a fluid drachm.

The prognosis in cases of poisoning with sulphuric acid is not good, about two-thirds of the cases proving fatal. Recovery has taken place after swallowing one fluid ounce of the strong acid, and one case is recorded of recovery after taking three ounces (by weight?) of the commercial sulphuric acid.

Fatal period.—The shortest time in which sulphuric acid has produced a fatal result is three-quarters of an hour (on this occasion three fluid ounces and a half of the concentrated acid were taken). The usual period in which sulphuric acid kills, if death result from the primary effects of the acid, is from eighteen to twenty-four hours. If death occur soon after taking the poison, it may be due to shock, to suffocation from swelling of the glottis, to pulmonary thrombosis or embolism due to the action of the acid on the blood, or to perforation of the stomach. Death may occur from exhaustion towards the end of the first week, on account of the inability to take or assimilate food. Finally death may occur weeks or months after the poison has been taken, either from destruction of the gastric glands, and consequent emaciation from inability to assimilate food, or from stricture of the œsophagus produced by the contraction of an œsophageal ulcer in the process of healing.

Analysis and tests.—To extract sulphuric acid from organic admixtures, such as the contents of the stomach, digestion of the organic substance with alcohol is required; on subsequent filtration the free sulphuric acid is found in the filtrate, the acid reaction of which may be determined by test-papers. The filtrate is then neutralised with caustic potash, evaporated to dryness, and the residue dissolved in distilled water acidulated with hydrochloric acid; to this liquid the tests for sulphuric acid are applied.

Free sulphuric acid cannot always be detected in the tissues of those who have succumbed to its poisonous action, on account of its absorption into the circulation, and its neutralisation by the sodium carbonate of the blood. The detection of sulphuric acid is based upon the recognition of an acid by its reddening effect upon blue litmus paper, and by the following tests :—

I. Sulphuric acid gives with solution of barium nitrate a white precipitate which is insoluble in strong hydrochloric acid.

II. Sulphuric acid gives with solution of lead nitrate a heavy white precipitate which is insoluble in nitric acid.

III. To detect sulphuric acid in stains on cloth or other fabrics, the stained portion should be cut out and digested in alcohol with gentle heat ; the filtered liquid, if sulphuric acid be present, will have an acid reaction, and, after dilution with water, will answer to the tests given above. Sulphuric acid may in this way be detected in clothing after the lapse of many years. Fresh sulphuric acid stains may be recognised by their dampness ; old stains by the destruction of the organic fabric.

Nitric Acid

Nitric acid is also known as aquafortis or red spirit of nitre. The symptoms produced by poisoning with this acid are very similar to those produced by sulphuric acid, but instead of blackening the tissues with which it comes in contact, nitric acid stains them a yellow colour. The fumes of nitric acid may also produce poisonous effects.

Symptoms.—If the acid be swallowed in a concentrated form, these come on immediately, and are as follows :—(i) Violent burning pain in the throat and œsophagus, extending down to the stomach ; the pain is severe, and the body may in consequence be bent or almost doubled up. (ii) Retching and vomiting ; the vomit contains blood, but the vomited matters are not so dark as in the case of sulphuric acid poisoning, and

the shreds of mucous membrane that may be vomited up are frequently of a yellowish colour. (iii) The lips, tongue, and mucous membrane of the mouth are excoriated, the tongue generally being of a citron-yellow colour. (iv) Great difficulty of breathing, as, on account of the fumes evolved from nitric acid, the air passages are more likely to be affected than in cases of sulphuric acid poisoning. Frequent rigors may also occur. (v) Obstinate constipation is generally present.

The dress of the patient should be carefully examined for the yellow stains produced by nitric acid.

Treatment and antidotes.—The same as in connection with sulphuric acid poisoning (see p. 121).

Post-mortem appearances.—The skin of the mouth and lips is stained an orange-yellow to a brown colour where contact with the acid has occurred, and there may be yellow spots upon the hands and neck, if any of the acid has been dropped on those parts. The mucous membrane of the œsophagus is softened, and is of a yellow or brownish colour; the mucous membrane of the stomach may be of a yellow-brown, or occasionally of a greenish colour, through the action of the nitric acid on the colouring matter of the bile; it is softened and easily detached. The stomach is on rare occasions perforated. In the duodenum similar or modified changes may be found.

Fatal dose.—The smallest quantity of nitric acid that has proved fatal is two fluid drachms. Recovery has taken place after half a fluid ounce of the strong acid has been taken.

Fatal period.—The shortest time in which nitric acid has proved fatal is one hour and three-quarters. Generally, death takes place in from twelve to twenty-four hours. In infants, however, if the acid be poured into the mouth, death may take place in a few minutes from suffocation, provided the nitric acid passes into the air-passages and occludes them by causing the mucous membrane to swell. A patient may recover from the immediate effects of nitric acid, and yet die weeks or months afterwards from emaciation, caused by the destruction of the

gastric glands and consequent inability to assimilate food. On one occasion a woman died from exhaustion eight months after having swallowed nitric acid.

Analysis and tests.—Nitric acid can be separated from organic admixture by dilution with distilled water and subsequent filtration. The detection of nitric acid is based on the acid reaction of the liquid to test-paper and on the following tests:—

I. To the solution contained in a test-tube two or three crystals of ferrous sulphate are added, and the tube shaken for a few seconds; strong sulphuric acid is then poured down the side of the tube so as to form a layer at the bottom; if nitric acid be present, a black or dark brown colour will appear at the junction of the two liquids.

II. To the solution add some copper turnings, apply a gentle heat, and then carefully add strong sulphuric acid, when, if nitric acid be present, a liberation of a colourless gas (nitrogen dioxide) occurs, which assumes a reddish-brown colour as it mixes with the air in the test-tube.

III. If a solution containing nitric acid be neutralised with potassium or sodium carbonate and evaporated to dryness, on adding to the residue when cold a few drops of strong sulphuric acid and then stirring in a crystal of brucine, a bright blood-red colour is produced.

IV. If to a liquid containing nitric acid an aqueous solution of diphenylamine be added, and some concentrated sulphuric acid gently poured down the side of the test-tube so as to form a layer at the bottom, a blue ring develops at the junction of the two liquids.

Poisoning by Nitric Acid Fumes

Death from the fumes of nitric acid has occurred in several instances, occasionally from exposure to and inhalation of, the fumes consequent on the breaking of a large vessel of nitric acid. In such cases no serious symptoms have been noticed

or experienced for an hour or more after the inhalation of the fumes, but generally within from two to a few hours, difficulty of breathing, and coughing occur, accompanied by a sense of tightness in the lower part of the throat. If death take place, it rapidly ensues, as a rule in from about eight to ten hours, the symptoms resembling those of capillary bronchitis or pneumonia. The cause of death is asphyxia from blocking of the bronchial tubes with the softened and swollen mucous membrane, and from effusion of blood-stained mucus; the asphyxia is, no doubt, accelerated by the paralysing action of the acid on the terminations of the vagi in the lungs, causing the respiratory reflex to be cut off.

Death from sulphuric and nitric acids.—In 1889 a man named Lipski was convicted of the murder of a woman, by pouring nitric acid down her throat whilst in bed. In reality, a mixture containing more sulphuric than nitric acid was employed. Such a mixture converts cellulose (wood and cotton) into nitro-cellulose, which substance was found by Stevenson in the stains on the deceased woman's linen, and also in wood cut from the floor of the room in which the murder was committed.

Hydrochloric Acid

Hydrochloric acid is also known as muriatic acid, spirit of salt, or commonly as spirits of salts.

Symptoms.—The symptoms are similar to those produced by sulphuric or nitric acid (see pp. 120, 124). Hydrochloric acid does not, however, stain the skin with which it comes in contact, and although it may produce darkening of the mucous membrane of the œsophagus and stomach from its action on the blood, yet it does not, as a rule, produce the amount of blackening that sulphuric acid does. Hydrochloric acid is less active as a corrosive than either sulphuric or nitric acid, but on account of the ready evolution of its acid fumes, it is more apt to attack the air-passages than either of the latter.

Treatment and antidotes.—The same as in connection with sulphuric acid poisoning (see p. 121).

Post-mortem appearances.—The mucous membrane of the mouth is white or grey where the acid has come in contact with it; the mucous membrane of the œsophagus and stomach is inflamed and dark. Perforation of the stomach may occur, but is exceptional.

Fatal dose.—The smallest fatal dose of hydrochloric acid is one fluid drachm, or a teaspoonful. Recovery has taken place after swallowing two fluid ounces of the acid.

Fatal period.—The shortest time in which hydrochloric acid has produced a fatal result is two hours; the average period is twenty-four hours. Death has occurred several weeks after the swallowing of hydrochloric acid, from emaciation consequent on the destruction of the gastric glands, and subsequent inability to assimilate food.

Analysis and tests.—Hydrochloric acid can be separated from organic admixture by distillation. The detection of hydrochloric acid is based upon the acid reaction of the liquid to test-paper, and on the following tests:—

I. Silver nitrate gives with hydrochloric acid a white curdy precipitate, which is insoluble in strong nitric acid, but is soluble in solution of ammonia.

II. If hydrochloric acid be warmed with manganese dioxide and strong sulphuric acid, chlorine is evolved, which may be recognised by its odour, and by the blue colour it imparts to potassium iodide and starch paper.

Oxalic Acid[1]

Oxalic acid is commonly used for cleansing and dyeing purposes, and also for bleaching straw hats, &c. It has been taken in mistake for Epsom salts. Oxalic acid is a powerful irritant poison, but it also acts as a depressant to the nervous system and heart.

[1] Oxalic acid is an organic irritant poison, but is placed among the inorganic corrosive poisons for the sake of convenience.

Symptoms.—(i) A burning acid taste comes on immediately or soon after swallowing the acid, with great pain in the stomach. (ii) Violent vomiting occurs immediately or within a few minutes, and the vomited matters as a rule contain blood. (iii) Cold sweats and collapse generally supervene; the respiration is gasping; the extremities are cyanosed, or the cyanosis may be general; the pulse is small and irregular. (iv) Spasms and convulsions may occur. If the poison be taken well diluted, the nervous symptoms are apt to be most prominent, and may even resemble narcotic rather than irritant poisoning.

Treatment and antidotes.—The stomach-pump should not be employed. The object of the treatment is to neutralise the acid as speedily as possible, and to render it insoluble; this is best effected by the administration of some form of calcium carbonate or chalk, which can generally be obtained in the form of *whiting*, kept in most houses for cleansing and polishing plate. About half an ounce to an ounce of this should be crushed up, mixed with half a tumberful of water, and administered to the patient. In the absence of this form of chalk, a portion of the ceiling or cornice of a room may be chipped off, crushed, and administered in a similar manner. In this way the oxalic acid is converted into the insoluble and inert calcium oxalate; a similar result may be brought about by the administration of a pint or more of saccharated lime water. After rendering the poison inert by the antidote, the further treatment should be directed to overcoming the collapse by the administration of brandy either hypodermically or in the form of enemata mixed with beef tea or milk; as a rule, on account of the irritated condition of the stomach, the brandy cannot be retained if administered by the mouth.

Post-mortem appearances.—These vary with the amount of acid taken and the strength of the solution of the acid. The mucous membrane of the tongue, mouth, pharynx, and œsophagus may be whitened, and the lower part of the œsophagus may occasionally be found congested. The mucous membrane of the œsophagus may be eroded, while that of the stomach

is generally more or less inflamed, but occasionally is pale and softened.

Fatal dose.—The smallest quantity of oxalic acid that has been known to produce a fatal result is sixty grains; this occurred in a boy aged sixteen years. When the dose is over half an ounce, death is commonly the result; recovery occurred in one case after taking an ounce and a quarter of the acid.

Fatal period.—In a large dose oxalic acid is a very energetic poison; the shortest time in which it has produced a fatal result is three minutes. Usually death occurs within from half an hour to an hour, although it has occasionally been delayed for several days.

Analysis and tests.—Oxalic acid may be separated from the contents of the stomach by partially drying them over a water-bath, and exhausting with hot alcohol containing a little hydrochloric acid; the alcoholic solution is filtered, evaporated to dryness, and the residue dissolved in water. The detection of oxalic acid is based upon the acid reaction of the solution to test-paper, and on the following tests:—

I. If a solution of oxalic acid be neutralised with ammonia, and calcium chloride added, a white precipitate of calcium oxalate is thrown down, which is insoluble in acetic acid, but soluble in hydrochloric acid.

II. If a solution of lead acetate be added to a solution of oxalic acid, a white precipitate of lead oxalate is thrown down, which is soluble in nitric acid.

Salt of Sorrel

Salt of sorrel, or *salts of lemon*, as it is commonly known, is an acid potassium oxalate, sometimes called the binoxalate of potash. It is used for straw bleaching, and also for removing ink stains and iron stains from linen. Its poisonous effects are similar to those of oxalic acid, but it is not quite so powerful an irritant as that acid, since it is one quarter neutralised with potash. Half an ounce has proved fatal to an adult

in eight minutes. The treatment, post-mortem appearances, and tests are the same as those described in connection with oxalic acid. It may be distinguished from oxalic acid either by testing for the potassium contained in it, or by incinerating some of the salt on a piece of platinum foil, when a white residue of potassium carbonate is left, whereas oxalic acid if incinerated leaves no residue.

Caustic Potash

Caustic potash, or potassium hydrate, is a corrosive poison which has a strong caustic action on the tissues, and produces by contact with them a soft and greasy mass. Potassium carbonate, or salt of tartar, has a similar action to caustic potash, though it is not so powerful a corrosive. *Soap lees*, used in the preparation of soft soap, contain caustic potash, and the poisonous effects of caustic potash have been produced by the swallowing of such lees.

Symptoms.—(i) During the act of swallowing a burning caustic taste is felt, and there is rapid excoriation of the mucous membrane of the mouth and œsophagus. (ii) A sensation of heat and pain is felt in the throat, œsophagus, and stomach. (iii) Vomiting usually occurs; the vomited matters consist of a blood-stained fluid which generally contains shreds of mucous membrane. (iv) Purging occurs in the majority of cases, and is frequently accompanied by tenesmus. (v) In a short time the lips, tongue, and throat become swollen and red, and the patient experiences great distress, with dyspnœa, and difficulty in getting rid of the swollen and detached mucous membrane. (vi) Collapse occurs, with a cold and clammy surface, and a small and feeble pulse.

Treatment and antidotes.—Neither the stomach-pump nor the stomach-tube should be used, on account of the danger of perforating the corroded œsophagus and stomach. The treatment should be directed to the immediate neutralisation of the alkali by means of acid drinks. The patient should be made

to drink either vinegar and water, or lemon juice mixed with water, or a weak solution of citric or tartaric acid. Either opium or a hypodermic injection of morphine should be given to relieve the pain, and stimulants employed to overcome the collapse; the preferable method of administering the stimulants is by hypodermic injection of ether or brandy. Demulcent drinks, such as linseed tea, almond mixture, and olive oil, should be given.

Post-mortem appearances.—The mucous membrane of the mouth, throat, œsophagus and stomach is softened, inflamed, and generally eroded in patches; the colour of the parts affected may be bright red or dark; the stomach may be perforated through the corrosive action of the poison.

Fatal dose.—The smallest recorded fatal dose of caustic potash is forty grains.

Fatal period.—The shortest time in which caustic potash has produced a fatal result is three hours. Death may occur weeks or months after taking the poison either from stricture of the œsophagus, or from emaciation due to the destruction of the gastric glands and the consequent inability to digest and assimilate food.

Tests.—Caustic potash may be detected by its alkalinity to test-paper, and by applying the reactions for potassium. These are:—

I. If a solution of caustic potash be acidified with hydrochloric acid, and a few drops of platinic chloride added to the mixture, which is then well stirred with a glass rod, a yellow crystalline precipitate of the platinum and potassium double chloride will be formed.

II. If a loop of platinum wire be dipped into a solution of caustic potash and then introduced into a bunsen-flame, a characteristic lavender tint will be imparted to the flame. If a sodium salt be also present, the yellow colour imparted by the sodium to the flame will entirely mask the lavender tint produced by the potassium salt, but if such a flame be viewed through a dark blue glass, the yellow sodium rays will be

absorbed by the blue, while the lavender-coloured rays produced by the potassium salt will pass through the glass unchanged.

Caustic Soda

Caustic soda, as a poison, causes symptoms precisely similar to those produced by caustic potash, and the description of the symptoms, treatment, antidotes and post-mortem appearances given in connection with the latter substance will apply equally well to caustic soda.

Tests.—Caustic soda may be recognised by its alkaline reaction to test-paper, and by the intense yellow colour communicated by it to the bunsen-flame.

Ammonia

Ammonia is a gas, but a strong solution of the gas in water, known as *solution of ammonia, liquid ammonia*, or *spirit of hartshorn*, is the common form in which this substance acts as a poison. A strong solution of ammonium carbonate may also exert poisonous effects. The vapour or gas may destroy life by setting up inflammation of the larynx, bronchi, or lungs.

Symptoms.—The symptoms are similar to those produced by caustic potash (see p. 131), but the combined sensation of heat and pain is much greater. The mucous membrane of the mouth, œsophagus, and stomach is quickly affected, and comes off in strips. Some of the gas or vapour is always drawn into the larynx and trachea, and produces a feeling of suffocation, which is increased by the swelling of the glottis, resulting from inflammation; the breathing is noisy, and there is dread of impending suffocation, with great distress in respiration, whilst at the same time the patient is greatly troubled in the attempts to get rid of the viscid mucus and detached membrane that accumulate in the mouth.

In a case recently recorded,[1] a man took by mistake a mouthful of strong solution of ammonia, but was quickly

[1] *Boston Med. and Surg. Jour.*, 1891.

treated with a suitable antidote, &c. On the day after swallowing the poison, headache and delirium came on with a temperature of 100·5°, the urine passed was diminished in quantity, was very albuminous, and contained blood, epithelial, and granular casts. The albuminuria completely disappeared in about three days. and recovery was rapid and complete.

Treatment and antidotes.—The same as in connection with caustic potash poisoning (see p. 131). Tracheotomy may be necessary to relieve the dyspnœa due to the inflammation and swelling of the glottis.

Post-mortem appearances.—The same as those of caustic potash (see p. 132).

Fatal dose.—The smallest quantity of solution of ammonia that has produced a fatal result is a teaspoonful, or one fluid drachm, of the strong solution.

Fatal period.—Death has occurred a few minutes after swallowing solution of ammonia, but the fatal result may be delayed for several hours.

Analysis and tests.—Ammonia can be separated from organic admixture by distillation with distilled water. If putrefaction has commenced in the organic matters, it is useless to test for ammonia, as it may then be the result of the putrefactive changes only. To the distillate, or to the ammonia gas as it passes over, the following tests may be applied :—

I. The odour of the gas constitutes a fairly delicate test.

II. Dense white fumes are produced on bringing a rod, moistened with strong hydrochloric acid, in contact with the gas.

III. Moistened red litmus paper is turned blue when brought in contact with ammonia gas.

IV. If some Nessler's reagent be added to a solution of ammonia, a reddish-brown colour or precipitate is produced, according to the amount of ammonia present.

IRRITANT POISONS

CHAPTER X

Poisoning by phosphorus—Chronic phosphorus poisoning—Poisoning by iodine, bromine, and chlorine—Boracic acid—Potassium salts—Barium salts—Magnesium salts.

Phosphorus

Phosphorus is a poison frequently contained in rat-pastes; the tips of lucifer matches also contain phosphorus. It is most commonly used for the purpose of committing suicide, the luminosity, smell, and taste being such as to generally prevent its use for homicidal purposes. There are two varieties of phosphorus, one of which is poisonous—namely, the ordinary yellow phosphorus—whilst the other variety, the red or amorphous phosphorus, is non-poisonous.

In the attempt to commit suicide with phosphorus, one of the phosphorus rat-pastes is generally taken spread upon bread. The ordinary phosphorus rat-paste contains yellow phosphorus, fat, sugar, flour, and prussian blue, the latter being added to colour it. A sixpenny pot of such paste was examined by Stevenson, and found to hold 157 grains of paste, which contained eight grains of phosphorus. A small pot such as can be bought for a penny may contain as much as four grains of phosphorus. The percentage of phosphorus in most of these rat-pastes is generally from three to four per cent.

A case of poisoning has been recorded by Leonard Hill,[1] in which the typical symptoms and post-mortem appearances

[1] *The Lancet*, 1890.

of phosphorus poisoning occurred, and which presumably was caused by inunction of rat-paste. The patient, a girl, previous to the occurrence of death, had most positively denied having taken any of the paste by the mouth, but at the inquest the interesting fact came out in evidence that the deceased girl had been giving a 'dark *séance*' to other girls, and had, just before the onset of the illness, rubbed the paste into her hands and face to produce blue flame.

Symptoms.—Phosphorus is an irritant, and the symptoms of irritant poisoning may come on a few minutes after taking it, although generally there is an interval of some hours. The occurrence of the symptoms is, in a number of cases, very peculiar and interesting, and, for convenience of description, may be divided into the following three stages:—

First stage.—(i) A garlic-like taste in the mouth. (ii) Pain and oppression in the region of the stomach; this pain is increased by pressure, whereas the pain of ordinary colic is relieved by pressure. (iii) Eructation of phosphorus vapours, with a peculiar odour of the breath; in some cases the breath has been seen luminous in the dark. The eructation of the vapours and their peculiar odour have been noticed four days after taking the poison. (iv) Vomiting, the vomited matters after a time being generally of a dark or tarry nature; the vomit may be luminous in the dark. (v) Intense thirst is generally complained of. (vi) Purging is not a common symptom, but it occurs in about a quarter of the cases. Death may occur from collapse in the course of a few hours, or the patient, if suitably treated, may recover. In a number of cases, however, the following complications or sequelæ result.

Second stage.—This is a stage of intermission of the symptoms. During this period, which may last from two to three days, or longer, the patient may suffer from some malaise, or feeling of indisposition. Usually, if the case is proceeding to a fatal termination, after a period of two, three, or four days, the symptoms of the third stage commence.

Third stage.—(i) Jaundice sets in and rapidly increases.

The jaundice is first noticed by the conjunctival membrane becoming yellow, and afterwards by the yellow tint of the skin. (ii) The liver in a number of cases becomes greatly enlarged, and the abdomen distended. (iii) Vomiting of altered blood frequently occurs, the vomited matters being black and grumous. (iv) Purging may occur, and the motions may contain blood. (v) Great prostration of strength. (vi) A general tendency to hæmorrhage is common, such as bleeding from the gums, nose, and in women from the vagina; subcutaneous and submucous hæmorrhages, producing purpuric spots, ecchymoses, and mottlings under the skin and mucous surfaces also occur. (vii) The surface becomes cold; the pulse is quickened and feeble; the urine is scanty, high-coloured, and generally contains bile-pigments, and occasionally albumen, bloodcolouring-matter, and casts. (viii) Coma supervenes, and death occurs in the majority of cases about the fifth or sixth day. In only a few cases has recovery taken place after the supervention of enlargement of the liver and jaundice from phosphorus poisoning; when this does occur the liver gradually returns to its normal size. In a few fatal cases a diminution in the size of the liver has been observed previous to the occurrence of death. In some instances the third stage is rendered prominent by the hæmorrhagic nature of the symptoms, the blood being occasionally poured out simultaneously from the nose, mouth, bladder, kidneys, and bowels; this hæmorrhagic form of phosphorus poisoning resembles scurvy in many of its characteristics.

Although death in the majority of cases occurs about the fifth or sixth day after taking the poison, yet cases have been recorded in which weeks have elapsed before the oncoming of the symptoms of the third stage, and the most remarkable instance of the delay of the symptoms of hepatic degeneration that has been recorded is one described by West,[1] in which a woman took a quantity of rat-poison about the size of a walnut spread on bread and butter; after treatment with emetics

[1] *The Lancet*, 1893.

and oil of turpentine, she was discharged from the hospital a few days later; nine weeks after taking the poison the symptoms of the third stage came on and proved rapidly fatal. The symptoms that occurred in this remarkable case nine weeks after taking the phosphorus were jaundice, enlargement of the liver, vomiting of a coffee-ground fluid, hæmorrhages from the gums, collapse, and coma.

In cases of phosphorus poisoning, the cause of the jaundice is no doubt due to degeneration of the liver cells produced by the action of the poison; the enlargement of the liver that occurs is partly due to fatty degeneration and partly to congestion. When jaundice in phosphorus poisoning has supervened, the system is then not only under the poisonous action of the phosphorus, but is also injuriously affected by the retention of the biliary secretion and other bodies in the blood. The symptoms closely resemble those of acute yellow atrophy of the liver. Leucin and tyrosin have been found in the urine in cases of phosphorus poisoning, indicating the interference with the chemical processes normally going on in the liver, and which result in the conversion of nitrogenous bodies into urea. A similar occurrence takes place in acute yellow atrophy of the liver.

Treatment and antidotes.—In cases of phosphorus poisoning the stomach-pump or stomach-tube should, if possible, be at once used, and the stomach thoroughly washed out. The administration of old or oxidised oil of turpentine is generally advised, forty minims being given in the form of an emulsion made with mucilage every fifteen minutes for the first hour, and afterwards three or four times a day until the patient is out of danger. The utility of oil of turpentine in cases of phosphorus poisoning has been very much questioned; at one time it was believed that its sole use was on account of the small quantity of peroxide of hydrogen contained in it, the function of which would be to oxidise the free phosphorus to phosphorous or phosphoric acid. From experiments made by Bush[1] of Dorpat on some of the

[1] *The Lancet*, 1892.

lower animals, it appears that turpentine has the power of hindering to a certain extent the toxic action of phosphorus, the explanation of its action being that it forms a compound analogous to terebinthino-phosphoric acid, which body is less toxic than the phosporus contained in it. Thornton[1] considers that permanganate of potash is the best antidote. It should be administered well diluted with water (·5 or 1 per cent. solution), and copious drinks of the diluted solution should be given before the poison has had time to become absorbed. Peroxide of hydrogen in the form of *sanitas* (a mixture of turpentine and water oxygenated by the prolonged action of air, and containing peroxide of hydrogen and camphoric acid in large quantities) may be given, in half-drachm doses, mixed with water; but it is somewhat slow in its action, and apt to further irritate the already irritated gastric mucous membrane. An emetic of sulphate of copper, which is frequently advised, should be avoided, as severe gastro-enteritis is liable to follow its employment. The further treatment consists in the administration of mucilaginous drinks, to which magnesia may be added, and the alleviation of pain by the use of opium or morphine if necessary. The administration of oils or fats should be avoided on account of their solvent action on phosphorus, and the consequent greater liability of the poison gaining access to the circulation.

Post-mortem appearances.—The stomach and intestines show signs of irritation, inflammation, and, in some cases, even of ulceration; the stomach in the majority of cases contains a liquid of a tarry colour or coffee-ground nature, due to changes in the blood that has been effused. In cases of recent poisoning the viscera may possess the peculiar odour of phosphorus, and even be luminous in the dark. Hæmorrhages are sometimes found in the pleural cavities, and, on rare occasions, on the surface of the brain. The most peculiar and characteristic post-mortem appearances of phosphorus poisoning are, however, those presented by the liver. In the majority of cases this

[1] *Therap. Gaz.*, 1893.

organ is considerably enlarged, although it may be found of a normal size, and on some occasions it has even been found contracted; it is of a pale yellow colour, sometimes firm in consistency, sometimes easily torn; small hæmorrhages are frequently present on the surface and in the substance of the liver. On microscopic examination the liver is found to be in a condition of acute granular or parenchymatous degeneration, a large amount of fat being present, the result of degeneration of the liver cells; the outline of the liver cells is in most places more or less destroyed, and the nuclei are almost invisible. The actual processes producing these degenerative changes in the liver cells are unknown, but they are probably the result of the blocking of the bile capillaries by swelling of the epithelial cells. The condition of the liver is very similar to that of acute yellow atrophy, with this difference—that in the majority of cases of phosphorus poisoning the liver is enlarged, whereas in cases of acute yellow atrophy the liver is diminished in size; but in those cases of phosphorus poisoning in which the liver has undergone advanced changes it cannot microscopically be distinguished from the liver of acute yellow atrophy. Poore [1] considers that, clinically and pathologically, the two conditions are indistinguishable. According to Hessler,[2] out of sixty-four cases of phosphorus poisoning, in thirteen the liver was small. The size of the liver is doubtless dependent on the period at which death occurs. If the patient die early, it may be large; if, on the other hand, the patient survive some time, it may be in a state of marked atrophy. On account of the strong resemblance of phosphorus poisoning and acute yellow atrophy of the liver, it is probable that the same pathological conditions exist in the two diseases. The hæmorrhages that so frequently occur in phosphorus poisoning are probably due to fatty degeneration of the vessel walls and to thromboses, the result of a tendency of the blood corpuscles to agglutinate together. The pleural and pericardial cavities frequently contain bloody serum, and bloody spots may be scattered over the pleura, pericardium,

[1] *The Lancet*, 1888. [2] *Vierteljahrsschr. f. ger. Med.*, Bd. 36.

and occasionally the endocardium. The blood is generally dark and fluid, and the corpuscles are more or less disintegrated. In a fatal case of phosphorus poisoning recorded by Elkins and Middlemas [1] certain changes were observed in the brain, as well as some small hæmorrhages on the surface; the grey matter of the cortex and the basal ganglia were of a rosy pink colour and congested appearance; in the cerebellum, pons, and medulla similar lesions were found to those present in the cortex of the brain; fatty changes were present in the walls of the larger capillaries, and the larger cortical nerve cells also showed fatty degeneration.

Fatal dose.—The smallest fatal dose recorded for an adult is one-eighth of a grain. The usual fatal dose is from one to two grains. The case of a woman is quoted by Galtier, who took in divided doses rather less than one grain of dissolved phosphorus, and death occurred in three days. The smallest fatal dose that has ever been recorded is one taken by an infant, five weeks of age, who died from sucking a single match-head, which probably contained about one-fiftieth of a grain of phosphorus. The action of phosphorus is more powerful if it be in a fine state of division, as in phosphorus paste or in lucifer matches, but it is still more powerful in its action if taken in a dissolved state. Recovery has taken place after between four and six grains of phosphorus in rat-paste have been taken.

Fatal period.—The shortest time in which death has occurred from phosphorus poisoning is half an hour; generally several days elapse before the fatal event occurs, the common period being about five to six days.

Analysis and tests.—In cases of suspected poisoning by phosphorus, the vomited matters, the contents of the stomach, and the viscera should be carefully smelt in order to notice whether any odour of phosphorus be evolved, and they should also be examined in the dark for the peculiar luminosity known as phosphorescence. Search should also be made in the vomited matters and in the contents of the stomach for particles

[1] *Brit. Med. Jour.*, 1891.

of prussian blue or other colouring matters with which the phosphorus may have been mixed. The most delicate test for phosphorus in its free state is the luminosity of its vapour when the phosphorus is expelled by boiling. The method is known as **Mitscherlich's test**, and is carried out in the following manner. The matters to be examined are placed in a flask, sufficient water is added to render the mixture fluid, and enough sulphuric acid to acidulate it; the flask is then attached to a condenser. The distillation is carried on in the dark, which is best effected by enclosing the condenser in a box, the inner walls of which are painted black; two eye-holes are provided in one of the walls, so that the condensing tube can be observed in perfect darkness. If phosphorus be present, the inner tube of the condenser becomes luminous either in part or throughout its entire length, according to the amount of phosphorus present. This is an extremely delicate test, and by its aid minute quantities of phosphorus may be detected, Another test known as **Scherer's test** is based on the reducing action of phosphorus vapour upon a solution of silver nitrate, which becomes blackened if exposed to its action. The best method of applying this test is to place some of the substance to be examined in a flask with some powdered lead acetate, which is employed to combine with and fix any sulphuretted hydrogen or other sulphur compounds that may be present. A little ether is added, and the mixture well shaken up; a slip of white filter paper, moistened with a solution of silver nitrate, is then suspended in the flask above the mixture, and the flask is set aside in a dark place to avoid the reducing effects of light upon the silver nitrate. At the end of an hour, if phosphorus be present, the paper will be found blackened from deposition of metallic silver.

If death has occurred several days after the taking of the poison, the phosphorus in such cases is generally oxidised to phosphoric acid, so that free phosphorus is not likely to be found in the stomach or its contents, or even in the viscera.

Chronic Phosphorus Poisoning

Chronic phosphorus poisoning is caused by the repeated inhalation of the vapour or fumes of phosphorus, and is especially liable to occur amongst those engaged in the manufacture of phosphorus matches. The bad effects of the phosphorus vapour are not, as a rule, immediately felt, although a certain amount of gastric discomfort and bronchial trouble may be complained of soon after commencing work, but frequently months and even years elapse before signs of illness appear. The first symptoms that are generally complained of are, weakness, griping pains or sensations of heat in the stomach, diarrhœa, wasting, and symptoms of bronchial catarrh. After a time toothache may come on, due to the phosphorus vapour setting up periostitis in the upper or lower jaw, which is generally caused by a carious tooth allowing the access of the phosphorus vapour or its acid to the periosteum beneath. As a result of this, the gum becomes swollen and inflamed, and separates from the alveolar process; the teeth in consequence become loosened or fall out, and pus of an offensive nature may issue from the diseased jaw. The bone in this way becomes exposed, and the periostitis frequently leads to necrosis. The general system suffers, in part from the action of the phosphorus, in part from the disordered nutrition, due to incapacity to properly masticate food, and in part from the swallowing of the offensive pus produced by the diseased jaw. The lower jaw is the one most frequently affected, no doubt because the saliva containing the dissolved phosphorous or phosphoric acid is brought in contact with it by gravitation.

The treatment of such a case is the removal of the patient from exposure to phosphorus fumes, and the surgical treatment of the affected jaw. If necrosed bone be present, it must be removed by operation. The prevention of this disease is only to be ensured, if common phosphorus be employed, by thorough ventilation and down drafts for the removal of the phosphorus

fumes, and the employment of workpeople with sound teeth, together with the periodical examination of their teeth.

Iodine

Iodine acts as a powerful irritant to the stomach and intestines. Cases of poisoning by iodine are most commonly due to the swallowing of the tincture or liniment by mistake. The colour and powerful smell of iodine prevent its being used for homicidal purposes.

Symptoms.—(i) A burning pain in the throat, mouth, and stomach. (ii) Vomiting and diarrhœa; the vomited matters either possess the yellow or brownish colour of the iodine, or, if starchy food be present in the stomach at the time of taking the poison, they are blue or black. (iii) Great thirst is generally complained of, with headache and a feeling of faintness. (iv) Symptoms of collapse occur, with coldness of the surface and a small feeble pulse.

Treatment and antidotes.—An emetic of warm water should be administered, and the stomach-tube employed to thoroughly wash out the stomach. Starch in the form of arrowroot, and demulcent drinks, such as linseed tea, barley-water, and milk, should be given.

Post-mortem appearances.—The mucous membrane of the œsophagus, stomach, and intestines is generally found inflamed and excoriated, and, as a rule, it is easily detachable from the muscular wall.

Fatal dose.—One fluid drachm of the tincture has caused death; this contains about one grain and a half of solid iodine. Recovery has taken place, however, after taking one fluid ounce of the tincture.

Fatal period.—Death has occurred in twenty-four hours. Only a few cases of fatal poisoning by iodine have been recorded.

Analysis and tests.—Iodine can be removed from organic admixture by agitation with carbon bisulphide

I. Iodine is readily detected by the dark blue colour that it forms with a cold decoction of starch.

II. A test for iodine in aqueous solution is to add chloroform, and well shake the mixture ; on standing, the chloroform settles to the bottom, carrying with it the iodine in solution, which imparts to it a purple or violet colour.

Bromine

Bromine is a liquid of a dark brown colour, possessing a most irritating odour. Only three fatal cases of poisoning by bromine have been recorded.

Symptoms.—(i) Intense burning pain in the throat and stomach, with eructations of bromine vapour. (ii) Collapse, which may produce a fatal result within a few hours of taking the poison.

Treatment and antidotes.—An emetic of warm water should be administered, and the stomach-tube used to thoroughly wash out the stomach. Starch, in the form of arrowroot, and demulcent drinks, such as linseed tea, barley water, and milk, should be given.

Post-mortem appearances.—The tongue may be considerably darkened in colour by the bromine. The mucous membrane of the œsophagus and stomach is generally considerably inflamed, and shows the presence of small submucous hæmorrhages. The mucous membrane is usually easily detached, and the walls of the stomach may be stained throughout by the bromine, or even be corroded where the bromine has lain for a long time in contact with the stomach wall.

Fatal dose.—Nothing definite is known of the quantity required to kill. Death has occurred after swallowing one ounce of bromine.[1] In a case recorded by Herwig,[2] in which a girl, aged ten, was given by a quack a mixture containing potassium bromide to be taken with chlorine water, death

[1] *New York Jour. of Med.*, 1850.
[2] *Zeitschr. f. Medicinalbeamte*, 1889.

occurred after taking three doses, which were ascertained to yield on the addition of chlorine about two grains of bromine.

Fatal period.—The shortest time known in which bromine has proved fatal is between seven and eight hours.

Analysis and tests.—Free bromine may be separated from organic admixture by distillation. Bromine may be recognised by its dark-brown colour and peculiar irritating smell. If an aqueous solution containing free bromine be agitated with chloroform and allowed to stand, the chloroform settles to the bottom, carrying with it the bromine in solution, which imparts a yellow or brownish colour to it. Bromine may also be recognised by the deep yellow colour that it imparts to a cold decoction of starch.

Chlorine

Chlorine is a yellowish-green gas with an extremely irritating smell. When inhaled, it sets up great irritation of the respiratory tract. The symptoms produced by the inhalation of chlorine are: severe coughing, difficulty of breathing, and pain in the chest. If death occur from the inhalation of chlorine, it is probably due to paralysis of the heart.

Boron

Boracic acid.—Fatal poisoning by boracic acid has occurred from washing out the stomach with that substance, and from the injection of a solution of it into the pleural sac and into an abscess cavity.

The symptoms are generally those of depression, with an erythematous or erysipelatous rash on the face and body. Vomiting and diarrhœa generally occur; blood may appear in the urine, and finally collapse may take place. The skin eruption is one of the most constant symptoms of boracic acid poisoning, and in cases of non-fatal poisoning by boracic acid, from its use in surgical practice, erythematous or urticarial eruptions have generally been noticed.

SALTS OF THE ALKALIS AND OF THE ALKALINE EARTHS

Potassium Nitrate

This salt is also known as nitrate of potash, saltpetre, or, if fused and moulded into small balls, it is known as Sal Prunella. It has been taken in mistake for Epsom salts and Glauber's salts, and in large doses it has on several occasions destroyed life.

Symptoms.—(i) Pain in the epigastrium followed by vomiting. (ii) Purging generally occurs, and the evacuations not unfrequently contain blood. (iii) Convulsions of the muscles of the face may occur. (iv) The pulse is weak and irregular. (v) Respiration is difficult. (vi) The extremities become cold and collapse occurs.

Treatment.—No antidote for nitrate of potash is known. The stomach-pump should be used to remove the salt and to wash out the stomach, or an emetic of mustard and water should be given. The after-treatment consists in the administration of mucilaginous drinks, and the employment of warmth and stimulants to overcome the collapse and the coldness of the extremities.

Post-mortem appearances.—The mucous membrane of the stomach is inflamed and easily detached, and in one case even perforation has been observed; blood-stained fluid may be present in the stomach; inflammation of the mucous membrane of the intestines may be found throughout.

Fatal dose.—The smallest fatal dose recorded is two drachms, which caused the death of an adult man. Recovery has, however, taken place after taking an ounce of the salt.

Fatal period.—Death does not, as a rule, occur for some hours after taking nitrate of potash, the shortest period that has been recorded being about two hours.

Tests.—The tests are those described for potash (see p. 132), and for nitric acid (see p. 126).

Potassium sulphate in large doses (two ounces) has acted as a poison, its effect being that of a combined irritant and depressant.

Potassium Chlorate

Potassium chlorate or chlorate of potash in large doses may act as a poison; its action is peculiar in that the red corpuscles of the blood are broken up by it, and the hæmoglobin is converted into methæmoglobin.

Symptoms.—(i) Signs of gastro-intestinal irritation with pain in the stomach and bowels, and vomiting. (ii) Cyanosis or pallor of the surface. (iii) Collapse. (iv) Jaundice frequently occurs after a time. (v) The urine is diminished in quantity or entirely suppressed—it may contain hæmoglobin. (vi) The patient may become somnolent or delirious, and death may occur after a few days from nephritis.

Treatment.—The stomach-tube should be used and the stomach thoroughly washed out. The after-treatment should be directed to the alleviation of any of the symptoms that may arise. If the kidneys are attacked, vapour baths and dry or wet cupping of the loins may be employed, and a mild diuretic, such as *liquor ammoniæ acetatis*, should be given.

Post-mortem appearances.—The mucous membrane of the stomach may be found swollen, softened, and easily detachable; small submucous extravasations of blood may be present. The blood presents a chocolate colour. The kidneys generally show a similar colour, with indications of acute nephritis. The spleen may be enlarged and also present, more or less, a chocolate colour.

Fatal dose.—An ounce and a half of chlorate of potash has proved fatal. The smallest fatal dose recorded is in the case of a child, three years old, who was killed by from forty-five to fifty grains.

Fatal period.—From a few hours to some days. The shortest time in which the salt has produced a fatal result is in the case of a woman who died five hours after swallowing it. If

death occur from nephritis, it does not take place for several days.

Analysis and tests.—Potassium chlorate may be separated from organic admixture by dialysis.

I. If a solution of potassium chlorate be coloured with a drop or two of solution of indigo sulphate, and a few drops of strong sulphuric acid added to the mixture, the blue colour of the indigo is bleached.

II. If potassium chlorate be obtained in the solid state, and a fragment of it warmed in a test-tube with a drop or two of strong sulphuric acid, a sharp explosion occurs, due to the decomposition by heat of the peroxide of chlorine evolved.

Potassium Iodide

Iodide of potassium has given rise to poisonous symptoms, even when administered in medicinal doses. With certain people the symptoms known as *iodism* are due to an idiosyncrasy, which causes them to be peculiarly susceptible to the action of this drug. The symptoms generally consist of headache, hot skin, discharge from the eyes and nostrils, inflamed conjunctivæ, dryness and irritation of the throat, and occasionally vomiting and purging. Erichsen has recorded a case in which alarming symptoms were produced by a dose of only five grains of iodide of potassium. The appearance presented by a person suffering from an attack of iodism may be such as to at first closely resemble an attack of erysipelas, but the temperature in all cases remains normal or subnormal.

Barium

The soluble salts of barium act as irritant, convulsant, and cardiac poisons. The chloride and nitrate of barium have both been taken in mistake for Epsom salts, Glauber's salts, and Carlsbad salts.

Symptoms.—(i) Pain in the stomach and abdomen. (ii) Vomiting and purging, with tenesmus. (iii) Thirst. (iv) Con-

vulsions. (v) Paralysis, the first indication of which is, as a rule, dilatation of the pupils.

Treatment and antidotes.—An ounce of sulphate of soda or Glauber's salts dissolved in a tumbler of water should be administered, with the object of converting the soluble barium salt into the insoluble sulphate of barium. The stomach-pump or tube should be afterwards employed, and the stomach thoroughly washed out. The after-treatment should be directed to overcoming the depressant and convulsant action of the poison. The patient should be wrapped in warm blankets, and hot-water bottles applied to the feet; brandy should be given either by the mouth, rectum, or hypodermically, to counteract collapse. If much pain persist, hypodermic injections of morphine should be employed.

Post-mortem appearances.—The mucous membrane of the stomach has been found very much swollen and red in places; in one instance the stomach was found perforated. Intense inflammation of the rectum has been frequently observed in fatal cases. The kidneys have been found congested, and the cavities of the heart generally contain a small amount of blood, partly coagulated and partly fluid.

Fatal dose.—The smallest quantity of a soluble barium salt that has produced a fatal result is one hundred grains, which caused the death of an adult woman in fifteen hours.

Fatal period.—One hour is the shortest time in which death has resulted from poisoning by barium salts, but the fatal period has been extended to seven days.

Analysis and tests.—Barium may be separated from organic admixture by drying, incinerating, and extracting the residue with nitric acid. The acid filtrate is evaporated to dryness, the residue dissolved in distilled water, and the following tests applied:—

I. Barium salts give with dilute sulphuric acid a white precipitate of barium sulphate, which is insoluble in either hydrochloric or nitric acid.

II. A solution of potassium chromate gives a yellow pre-

cipitate of barium chromate with barium salts. This precipitate is insoluble in acetic acid, but is soluble in either hydrochloric or nitric acid.

III. If a barium salt be introduced into a bunsen-flame on a coil or loop of platinum wire, previously moistened with hydrochloric acid, a greenish colour is imparted to the flame.

Magnesium

Magnesium sulphate or Epsom salts has in large doses caused death; although it is generally regarded as a harmless purgative, yet cases have occurred in which, especially when taken upon an empty stomach, a fatal result has been produced. The author in 1887 investigated a case of death in a young woman aged twenty, which, from the results of the enquiry, appeared to be due to poisoning by Epsom salts. The deceased had retired to her bedroom at night apparently in her usual health, and the following morning was found lying on her bed fully dressed and dead. The bed had not been occupied, and death had occurred some hours previously. On the dressing-table was a tumbler containing some small white crystals, which on examination were found to consist of Epsom salts only. The stomach was not inflamed, and contained an ounce and a half of a light-coloured semi-fluid mixture, in which analysis revealed only the presence of Epsom salts as an abnormal constituent. It came out in evidence that the deceased had purchased, the day before her death, an ounce of Epsom salts, which she had apparently taken on an empty stomach, with the result that death probably occurred from fatal syncope.

A case of poisoning by Epsom salts is also recorded by Sang[1]; a woman, thirty-five years of age, purchased four ounces of Epsom salts, and took the whole quantity in a tumbler of hot water. Soon after she complained of a burning pain in the stomach and bowels, with difficulty of breathing

[1] *The Lancet*, 1891.

or a choking sensation. In about one hour from taking the Epsom salts she was in a state of hopeless collapse: the pupils were dilated; there was a slight twitching of the muscles of the face, and complete paralysis. She rapidly became comatose, and died about an hour and fifteen minutes after taking the dose. Christison, in his work on poisons, mentions the case of a boy ten years old who took two ounces of Epsom salts partly dissolved and partly stirred up in a cupful of water. Directly after taking it he was observed to stagger and become unwell. When seen by a surgeon half an hour later the pulse was imperceptible, the breathing slow and difficult, the whole frame in a state of extreme debility, and in ten minutes more the child died without any vomiting.

CHAPTER XI

Poisoning by arsenic—Acute arsenical poisoning—Chronic arsenical poisoning—Substances containing arsenic or its compounds—Extraction of arsenic from viscera—Marsh's test—Reinsch's test—Estimation of arsenic.

Arsenic

The white arsenic of commerce is arsenious oxide or arsenious anhydride. It occurs in two varieties—a crystalline variety which can be obtained in small octahedral crystals, and a vitreous variety constituting the deposit in the condensing flues attached to the furnaces in which the arsenical ores are roasted. The vitreous variety is obtained at first as a semi-transparent glass-like solid, and becomes, after a time, opaque like porcelain. The white arsenic of commerce is obtained by grinding this vitreous variety to a powder. White arsenic is a white, odourless, and almost tasteless substance, possessing a very faint sweetish taste. It is very poisonous, and it is on account of its practically tasteless character that it may be successfully given for homicidal purposes in food or medicine, since it is swallowed by the victim without its presence being detected by taste. The question as to the extent of the solubility of arsenic is one that may arise in the judicial investigation of cases of poisoning by this substance. In cold water from half to one grain of arsenic dissolves in a fluid ounce, but in boiling water from six to twelve grains are carried into solution, the degree of solubility varying according to the time that the arsenic has been kept, and its consequent molecular condition. A peculiarity of powdered arsenic when mixed with water is the way in which it floats on the surface, forming a white scum, which

rapidly forms again on the surface even after thorough agitation. If arsenic be sold to the public in quantities of less than ten pounds, the law requires that it should be mixed with soot or indigo to colour it, from one-tenth to one-sixteenth part of its weight of one of these colouring substances being employed. With regard to the substances in common use that contain or may contain some preparation of arsenic, see pp. 163–165.

Symptoms.—The symptoms of poisoning by arsenic in acute cases generally come on from half an hour to an hour after taking the poison. On one occasion they appeared almost immediately. The rapidity with which they appear depends very much as to whether the arsenic is taken in solution or not, and as to whether the stomach is empty or not at the time. If taken in solution, the symptoms appear much more quickly than if taken in a solid form, and if taken upon an empty stomach, they are more rapid in their occurrence than if taken upon one containing food. The maximum period to which the appearance of symptoms of acute arsenical poisoning has been protracted is ten hours.

The following are the symptoms of acute arsenical poisoning:—(i) An intense burning pain in the throat and stomach generally occurs, and this pain is usually increased by pressure; on a few occasions the pain has been entirely absent. (ii) Faintness, nausea, and sickness generally supervene. (iii) Intense thirst is a very common symptom, but occasionally is absent. (iv) Violent vomiting generally occurs, the vomited matters at first consisting of food, if any be present in the stomach, and later on of mucus, which is sometimes streaked with blood, or is bile-stained, or may even resemble rice water in appearance. (v) Purging with tenesmus, the discharges from the bowels being frequently tinged with blood. If the diarrhœa continue the discharges from the bowels may resemble rice water in appearance, and may exactly simulate the stools that are seen in typical cases of Asiatic cholera. (vi) The pulse is small, rapid, and irregular. (vii) Collapse generally occurs, with a cold and clammy skin. (viii) Severe

cramps in the calves of the legs. (ix) When a fatal termination is approaching the respiration generally becomes laboured. (x) Restlessness is common; usually stupor with convulsions and spasms, or accompanied with paralysis, supervene shortly before death. In a few cases of acute arsenical poisoning, the poison appears to especially affect the nervous system, and in this form—the nervous form—vomiting and purging may be either entirely suppressed or only present to a slight degree, symptoms due to affection of the nervous system being most prominent, such as stupor, paralysis, delirium, convulsions, and even acute mania.

The course of the symptoms produced by arsenical poisoning may be considerably modified if repeated doses of the poison are given, as is not uncommonly the case when the poison is administered for homicidal purposes. In addition to the symptoms of acute arsenical poisoning, such as pain in the stomach, sickness, purging, loss of appetite, and feeling of depression, some of the symptoms of chronic arsenical poisoning may supervene later on. These are especially injection of the conjunctivæ, smarting of the eyelids and eyeballs, irritation of the mucous membrane of the fauces and throat, and dryness of the tongue and mouth; the tongue is red, coated and irritable looking; the skin presents a sallow or partially jaundiced appearance, and eruptions of an erythematous or eczematous nature frequently appear. In addition to these symptoms, signs of peripheral neuritis may occur, such as a feeling of formication or tingling in the fingers, with numbness of the extremities, muscular cramps, and tenderness of the muscles on pressure.

A brief account of the Maybrick case, in connection with which Mrs. Maybrick was tried and convicted at the Liverpool Assizes, 1889, will be of interest as representing the symptoms which arose from repeated doses of arsenic administered during a period of probably about fourteen days. On April 27th Mr. Maybrick was seized with vomiting which came on after taking tea. On the following day he still suffered from vomiting, with

foulness of the tongue, and also complained of stiffness in the lower limbs. On May 1st he complained of feeling unwell after taking luncheon, and on the three following days he was sick, and complained of a tickling sensation in the throat accompanied by retching. On May 7th he was still suffering from vomiting. Diarrhœa was then appearing, and the throat was very dry and acutely inflamed. On May 8th he was suffering from diarrhœa with considerable straining. On May 9th distressing tenesmus was present, and on May 11th he died.

Cases of acute poisoning by arsenic may be suitably treated so that the bulk of the poison is removed from the system, and yet severe symptoms may occur later on. The following cases are of interest as illustrating this point. Jolly [1] relates a case of attempted suicide by a woman aged twenty-seven. After the gastro-enteritis had passed away, loss of sensation was noticed in the hands and feet about the fifth day; loss of muscular power afterwards appeared, and by the fourth week she was unable to walk unaided; paresis and atrophy of the muscles became well marked, and the reaction of degeneration appeared in some of the affected muscles. The loss of muscular power and sensation was due to peripheral neuritis. Kovacs [2] records a case in which a man, forty years of age, took 150 grains of white arsenic. Symptoms due to severe gastro-enteritis were subdued by appropriate treatment, but a week later symptoms of peripheral neuritis developed. There were first œdema and coldness of both lower extremities, and a week later some anæsthesia of the feet, lightning pains, an unsteady gait, and anæsthesia began to make its appearance in the finger tips and upper extremities. In some cases the latent period between the time of taking the arsenic and the development of the nervous symptoms has extended to even a longer time than in this case. As much as four weeks elapsed in a case recorded by Seeligmüller. These cases indicate the caution of not giving too early a favourable prognosis in cases of acute arsenical poisoning, even when the acute symptoms have

[1] *Deut. Med. Woch.*, 1893. [2] *Wiener Med. Wochensch.*, 1890.

entirely passed away. In cases of somewhat slow or chronic poisoning by arsenic, it should be carefully borne in mind that the symptoms very much resemble those which occur in connection with ordinary chronic gastritis, and with ulceration of the stomach.

The question is sometimes raised as to whether arsenic is an accumulative poison—that is, whether, if taken in a medicinal form for a prolonged period, it can accumulate in the body, and then suddenly give rise to the symptoms of acute arsenical poisoning. There are no facts to show that arsenic in any sense is an accumulative poison, and long experience has shown that in medicinal doses it may be given for a prolonged period without doing harm, owing to its rapid elimination by the kidneys in the urine, and to a slighter extent in the other secretions. Arsenic is for a short time deposited in the various organs, but it is rapidly removed from them, and even if a large dose of arsenic has been taken and the person survives, it may in the course of two or three weeks be entirely removed from the system, although it has on one occasion been detected in the urine forty days after its administration ceased, and in another case recorded by Putnam,[1] where arsenic had been given in medicinal doses, it did not disappear from the urine for nearly fifty days. Brouardel and Pouchet found, as a result of the investigation of several cases of wholesale poisoning that took place at Hyères and Havre in 1889, that arsenic could be found in the bones, and especially in the spongy tissue of the bones, when no trace of it was discoverable in the viscera. From experiments made at the same time on dogs and rabbits after the administration of arsenic, it was found that all traces of arsenic disappeared from the viscera in three weeks, but could be found in the bones ten weeks after the last dose had been given. Gibb,[2] however, records a case in which arsenic was found in the liver and bones six months after cessation of a long course of medicinal treatment.

The susceptibility of some persons to a small dose of

[1] *Boston Med. and Surg. Jour.*, 1888. [2] *Lond. Path. Soc. Trans.*, 1888.

arsenic should be remembered. Nicholson[1] relates the case of a patient who had been ordered three minims of *liquor arsenicalis* three times a day after food, to be taken well diluted. After taking five doses of the mixture, or fifteen minims in all of the *liquor*, a universal erythematous rash came out, the chest and abdomen presenting a boiled-lobster appearance. The patient was also attacked with some diarrhœa and suffusion of the conjunctivæ. The rash disappeared when the arsenic was discontinued. The dose of arsenic taken over a period of nearly two days that produced these symptoms was only from one-sixth to one-seventh of a grain.

Treatment and antidotes.—In cases of acute poisoning by arsenic, the stomach should be emptied by means of the stomach-pump or stomach-tube as speedily as possible, and then thoroughly washed out. In the event of neither the pump nor the tube being obtainable, an emetic of mustard and water should be given. The employment of freshly precipitated ferric hydrate or dialysed iron has been recommended as an antidote, with the object of converting the arsenic into an insoluble arseniate of iron. If employed, the ferric hydrate is best prepared by mixing half a fluid ounce of the tincture of perchloride of iron, diluted with half a tumblerful of water, with an equal quantity of a solution of washing soda, the latter being used in excess; the ferric hydrate so precipitated should be stirred up with the contents of the tumbler, and administered to the patient. In place of the ferric hydrate, drachm doses of the solution of dialysed iron, diluted with water, may be administered. It is doubtful, however, whether these iron preparations are of much value as antidotes in cases of arsenical poisoning; the early evacuation and washing out of the stomach is the preferable mode of treatment. For the collapse resulting from acute poisoning by arsenic, stimulants should be employed, brandy being given by the mouth if it can be retained, or in the form of enemata if diarrhœa be not present, or by hypodermic injection. For the relief of the nervous sym-

[1] *The Lancet*, 1893.

ptoms, subcutaneous injections of morphine should be employed, and if the diarrhœa and tenesmus are urgent, opium enemata may be administered. Demulcent drinks should be given to allay the gastro-intestinal irritation.

Post-mortem appearances.—These are mainly confined to the stomach and intestines. The whole mucous membrane of the stomach may be red and inflamed, the surface resembling in appearance red velvet, or the redness may be distributed in the form of streaks or dots of a dark red colour upon an inflamed mucous membrane of a lighter red; the dots are called petechiæ. These dark-coloured dots and streaks are not always present, and have been found absent in some cases in which the arsenic was administered in a soluble form; possibly their occasional presence may be due to the extreme irritation induced by particles of solid arsenic. The redness is especially evident on the prominences of the folds of the gastric mucous membrane. Submucous effusions of blood may be present, and the stomach frequently contains dark-coloured mucus tinged with blood. The gastric mucous membrane is occasionally found ulcerated or eroded, and on rare occasions has been seen in a gangrenous condition. A few instances are on record of perforation of the coats of the stomach as a result of arsenical poisoning, but they are very exceptional cases. If putrefaction has advanced in the stomach, the white arsenic may have been converted into the yellow sulphide, owing to evolution of sulphuretted hydrogen as one of the products of putrefaction; this sulphide of arsenic may colour the stomach throughout its coatings and appear as a yellow stain upon the peritoneal suface. The formation of the yellow sulphide of arsenic has also been observed upon the surface of the liver, on the omentum, and on the right side of the heart, and in a case of arsenical poisoning described by Murray,[1] the endocardium of the left ventricle presented a bright yellow stain or deposit over one-third of its area, which stain was proved by analysis to be the yellow sulphide of arsenic.

[1] *The Lancet*, 1892.

A yellow pigment has also been observed in the intestines of some bodies exhumed after arsenical poisoning. This may be due to the formation of the sulphide of arsenic, or to a peculiar alteration in the pigments of the bile. The intestines may show inflammation throughout, but this appearance is frequently only to be observed in the duodenum. Of the large intestines the rectum is the part most prone to inflammation. It should, however, be remembered that death may occur from acute arsenical poisoning, and large quantities of arsenic may be found in the stomach and the intestines, and yet no appearance of inflammation be present in those organs; so that the non-existence of inflammatory changes in the alimentary canal after death is not necessarily a proof that the individual has not died from the effects of arsenical poisoning.

Arsenic in many cases exercises a preservative action on the bodies of persons dying from acute arsenical poisoning, but such preservative action does not necessarily occur; it is only in the somewhat exceptional cases in which large quantities of arsenic have been taken that such instances of delayed putrefaction have been observed. The preservative effect in these cases is especially manifested in the internal viscera; in the case previously mentioned, described by Murray (p. 159), the viscera presented a fresh and well-preserved appearance, and there was an absence from them of the usual foul putrid smell. The time that had elapsed since death occurred in this case is not, however, given. In the case of a body exhumed five months after death, and examined by Brouardel and Pouchet, and which was a case of arsenical poisoning in which a considerable amount of the poison was found in the body, the viscera were discovered to be in a remarkable state of preservation, no gases of putrefaction being present. In the case of the exhumation of the body of a Mrs. Cross seven weeks after death (*Reg.* v. *Cross*, Munster Ass., 1887), death having been caused by arsenical poisoning, Pearson, who examined the body, states that all the organs were in a well-preserved state,

the stomach and intestines appearing as fresh as though the deceased had died twenty-four hours previously. An idea has been prevalent that the corpse of a person poisoned by arsenic undergoes a kind of mummification; the subject has been investigated by Zaaijer,[1] whose observations point to the conclusion that the so-called 'arsenical mummification' has no existence, and that the mummification of a corpse has no value as a judicial proof of poisoning.

Arsenic is not a normal constituent of the body, and, therefore, if found in the tissues after death, its presence must be due either to the poison having been taken by, or administered to, the deceased, or to its having been introduced into the body after death, as in some methods of embalming, or as in the method of preservation for the purposes of dissection by the injection of arsenical paste into the vessels. In connection with the discovery of arsenic in exhumed bodies, it has been suggested that its presence may be due to the passage of arsenic from the soil of the cemetery or graveyard into the tissues of the body. Arsenic has been found in minute quantities in the soil of some graveyards, but always in an insoluble form, and generally in combination with iron in the form of arsenical iron pyrites; so that it is extremely unlikely that a body buried in a cemetery or graveyard can become contaminated with arsenic derived from the soil. In cases of exhumation, it is advisable to collect a sample of the soil from around the coffin for the purposes of analysis. Arsenic has been detected in the body many years after death. It has been discovered by Glover in a body after twelve years', and by Webster in the remains of a body after fourteen years' burial.

Fatal dose.—The smallest quantity of arsenic that has proved fatal to an adult is two grains, but recovery has taken place after much larger doses.

Fatal period. The shortest period in which arsenic has proved fatal is twenty minutes, in which case a large dose of

[1] *Archives Neerland. des Sciences Exactes et Naturelles*, vol. xxi. part 5.

poison had been taken. The average time for a fatal result to occur in cases of acute arsenical poisoning is about twenty-four hours, but it may be much later. Mr. Maybrick probably died on about the fourteenth day from the commencement of the administration of arsenic.

External application of arsenic.—Death has occurred on several occasions from the external application of arsenic; in such cases arsenic is found in the viscera, including the stomach; the arsenic is carried to the different viscera dissolved in the blood, and may pass into the contents of the stomach by a process of transudation or osmosis. In a case in which an ointment containing arsenic and white precipitate was applied to the diseased head of a child, death occurred after ten days, and at the post-mortem examination the mucous membrane of the stomach and intestines was found red and inflamed, and traces of arsenic were discovered in the fluid contents of the stomach, in the intestines, and in the liver. This transference of absorbed arsenic from the blood to the stomach and intestines has been distinctly proved by the experiments of Taylor and Pavy.[1] Plasters containing arsenic have been applied by quacks to cancerous and other tumours, and have produced death as a result of the absorption of the arsenic by the diseased surface. In 1878 a number of children in Essex were killed from the employment of a dusting powder, supposed to be violet powder, which was dusted on their skins after they had been washed. This powder was subsequently found to contain one-third of its weight of arsenic. From one of the bodies Tidy extracted six grains and a half of arsenic.

Chronic Arsenical Poisoning

If very small quantities of arsenic are taken into the system for a prolonged period of time, symptoms of chronic arsenical poisoning may supervene, and these are somewhat different to

[1] *Guy's Hospital Reports*, 1860.

those of the acute form. Before describing these symptoms it will be as well to enumerate the various substances, or preparations, which contain or may contain arsenic, and which may give rise to chronic arsenical poisoning.

Preparations, substances, &c. containing arsenic or its compounds, which may give rise to arsenical poisoning:—

Metallic arsenic.—A preparation known as fly-powder consists chiefly of powdered metallic arsenic, probably mixed with some white arsenic, and is a strongly poisonous substance.

Compounds, preparations &c. containing white arsenic (As_2O_3), *or arsenious acid* (H_3AsO_3).—Arsenic was formerly put into candles to harden them, and to give them a wax-like appearance. During the burning of such candles, the arsenic becomes slowly volatilised, and can produce very dangerous effects. Arsenical solution, consisting of arsenic, soft soap, and tar, is frequently used by shepherds as a 'sheep-wash,' or 'sheep-dip,' for the purpose of destroying tick and other parasites in the wool of the sheep; bad effects have been produced in those engaged in washing sheep with such a solution. Arsenical weed killers are also in use for the purpose of destroying weeds. An arsenical soap is used for preserving the skins of birds and animals, and a powder consisting of a mixture of arsenic and plaster of Paris is used for a similar purpose. Persons engaged in rubbing these preparations into skins have been attacked by arsenical poisoning, and symptoms have even resulted from the keeping of stuffed birds or animals, whose skins have been so preserved, in dwelling-rooms. A solution of arsenic in carbonate of soda, prepared by dissolving in water a mixture of forty parts of white arsenic to nine parts of carbonate of soda, has been employed for cleansing steam boilers, such a preparation being a most dangerous one for the purpose. Arsenic has also been employed for facing, or producing a glaze upon, fancy paper, note-paper, cardboard boxes, playing cards, and wrappers for sweetmeats. Wheat is sometimes steeped in an arsenical solution, for the purpose of preserving the grain for seed, and this has been the cause of accidental poisoning both

to men and animals. Arsenic is also employed in glass-making and in white enamelling. It is sometimes given by grooms to horses, with the idea that it renders their coats glossy. It is a constituent of some of the rat pastes used for the destruction of rats. Arsenic has been detected in the glazed leather-linings of hats, and in some cases has produced a local eczema on the foreheads of persons wearing such hats. The ordinary silk coat-sleeve lining, the ordinary brown-paper carpet lining, and the common black cambric furniture lining have been found to contain arsenic. Some of the varieties of 'fly-papers' consist of paper soaked in a solution of sodium or potassium arsenite containing some sugar; the paper known as *papier moure*, which is used for killing flies, is so prepared. Stevenson has found some of the fly-papers of commerce to contain from seven and a half to ten grains each of arsenious acid in a soluble form; by preparing infusions of such papers, a strong solution of arsenic can be obtained, and such solutions have been used for criminal purposes. Hills[1] considers that coal and coal-gas are possible sources of arsenical poisoning in dwelling-houses.

Copper arsenite ($CuHAsO_3$).—This substance, known as *Scheele's green*, which possesses a bright green colour, has been extensively used as a pigment, and was at one time especially employed in the preparation of green wall-papers; but, owing to the dangerous effects that have resulted from the use of such wall-papers in rooms, the better class now manufactured are kept rigorously free from this substance. Scheele's green has also been employed as a green oil paint and in green water-colour paints, and also as a colouring agent for book-covers, lamp-shades, artificial flowers and leaves, green wax candles, wax tapers, india-rubber balls and dolls, japanned goods, carpets, floor-cloth, linoleum, printed calicoes, curtains, table-cloths, and fabrics such as cretonnes, chintzes, and green baize. Numerous cases of chronic arsenical poisoning have resulted from the presence in rooms of wall-

[1] *Boston Med. and Surg. Jour.*, 1894.

papers and other articles containing Scheele's green. *Emerald green* and *Schweinfurt green* consist of a mixture of copper arsenite and copper acetate, and have also been used for similar purposes to Scheele's green.

Orpiment, or *arsenious sulphide* (As_2S_3).—This body, also known as *yellow arsenic*, or *King's yellow*, is of a bright yellow colour, and is occasionally used in painting, staining, and colouring toys. When pure it is said to be non-poisonous, but the commercial variety is certainly poisonous owing to the presence of a varying amount of white arsenic.

Arsenic acid (H_3AsO_4).—This acid, in combination with potassium or sodium, as potassium or sodium arseniate, is used as a fly-poison. Arsenic acid is employed in the manufacture of some of the aniline dyes, and, if not completely removed from such dyes, it may be present in articles dyed with them. Injurious effects, such as local eczematous eruptions, have followed the wearing of socks and gloves coloured with such dyes containing arsenic, and also from the employment of cretonnes and other hangings coloured with arsenical dyes. Red aniline dyes are also occasionally used to give a colour to liqueurs, syrups, sweetmeats, and raspberry vinegar, and it is possible that injurious effects might result from the taking of such coloured articles, if the dye contained arsenic.

Arseniuretted or *arsenetted hydrogen* (AsH_3).—This body is a gas, and is the most poisonous compound of arsenic. It is produced whenever hydrogen is generated in contact with a soluble arsenical compound. It may be evolved in the bronzing of brass, in the tinning of sheet iron, and in the removal of silver from argentiferous zinc by the action of acid. It has produced poisonous effects in workmen exposed to its toxic action.

Symptoms of chronic arsenical poisoning.—If arsenic be taken into the system in repeated small quantities through the lungs, and possibly to some extent swallowed in saliva, as in the case of people living in rooms the walls and articles of furniture of which are contaminated with arsenical preparations,

some of the following symptoms may occur :—(i) Loss of appetite, and general malaise. (ii) Headache and neuralgia. (iii) Nausea, accompanied by gastric pain. (iv) Irritation of the throat, eyes, and nostrils ; the conjunctiva may be inflamed, and intolerance of light may be experienced. (v) Colic, constipation, or diarrhœa may be present. (vi) Gradual prostration of strength. (vii) Wasting, accompanied by anæmia, or by a peculiar cachectic hue of the skin. (viii) Spots of pigmentation may be present on the skin. (ix) Symptoms of peripheral or multiple neuritis may develop ; the symptoms are sensory, motor, and reflex, and closely resemble those occurring in connection with alcoholic neuritis. The sensory symptoms commence, as a rule, with tingling and numbness of the hands and feet, extending to the arms and legs ; hyperæsthesia of the muscles of the arms and legs follows, so that the slightest pressure may give rise to intense pain ; followed by, or accompanied by, cutaneous anæsthesia in some cases. The motor symptoms show themselves by paralyses, which especially affect the muscles concerned in the execution of the finer movements of the hands and feet ; in advanced cases there is inability to use the hands or to walk properly. These motor symptoms are generally attended with atrophy of the affected muscles and the development of the reaction of degeneration in them. The reflex symptoms are shown by absence of the knee-jerks and of the superficial reflexes. Symptoms of arsenical neuritis may result from the long continuance of full therapeutic doses of arsenic, as occurred in a case related by Osler.[1]

Brouardel recognises four distinct periods in arsenical poisoning. The first is marked by digestive troubles, the second presents eruptions, and catarrh of the larynx and bronchial tubes, the third is characterised by sensory troubles, the fourth period by paralysis.

In cases of peripheral neuritis of doubtful origin, it is advisable to carefully examine the urine for arsenic. In many

[1] *Montreal Med. Jour.*, 1893.

cases of chronic arsenical poisoning the cause has not been suspected until all ordinary treatment has failed, and until a chemical examination of the wall-paper, or of other articles previously enumerated, has revealed the presence of arsenic.

With regard to the modes by which arsenic contained in wall-papers in the form of Scheele's green can affect persons inhabiting rooms, the walls of which are covered with such papers, the following are the probable explanations: (i) Some of the arsenite of copper may be mechanically brushed off the walls in the form of dust, and so gain access to the lungs. (ii) Minute quantities of arseniuretted hydrogen may be diffused into the atmosphere of the room, on account of the paper being fastened to the walls with starch paste, which readily ferments and sets free nascent hydrogen; since the paper is originally saturated with the paste, this nascent hydrogen is produced in direct contact with the arsenical compound, and so forms gaseous arseniuretted hydrogen, which escapes into the air of the room. It is true that the quantity of gas so produced is very minute, but as arseniuretted hydrogen is the most poisonous of all the arsenical compounds, very small quantities may be sufficient to produce very injurious effects. (iii) From recent researches made by Gosio in the Public Health Laboratories at Rome, as to the influence of microphytes on arsenical wall-paper, it is found that, if certain moulds and microbes are cultivated and allowed to grow in contact with arsenical preparations, arseniuretted hydrogen is produced. The moulds seem to be the more active in effecting this transformation, and the *mucor mucedo* seems to be the most active. Apparently the process consists in the production, by the growing moulds, of nascent hydrogen, which, in contact with the arsenical compound, forms arseniuretted hydrogen.

Symptoms of poisoning by arseniuretted or arsenetted hydrogen.—This gas is the most poisonous of the arsenical compounds, and as it produces symptoms different to those of acute arsenical poisoning, a special description of them is necessary.

The severity of the symptoms of poisoning by arseniuretted hydrogen appear to be due, in part, at all events, to the rapid destruction of the red blood corpuscles by the gas. As a rule, from a few minutes to one or two hours after the inhalation of the gas, giddiness, vomiting, and shivering come on, followed by great prostration of strength, and generally accompanied by partial or complete suppression of urine. Death occurs from five to twenty-four days after the inhalation. A case of fatal poisoning by this gas occurred in the latter part of 1892, the victim being Dr. Schultze, Professor of Chemistry in the University of Santiago. In his case the red blood corpuscles were diminished, previous to the occurrence of death, to 1,800,000 per cubic millimetre in place of the normal 5,000,000 per cubic millimetre. Progressive uræmia followed, and death occurred in five days. The tissues were found at the post-mortem examination in a state of fatty degeneration. In the majority of cases of poisoning by this gas, so small a quantity has been inhaled, that its odour has not been recognised by the persons breathing it.

Analysis and tests.—Arsenic can be detected by some of the following tests in very minute quantities. As it is not a normal constituent of any of the tissues of the body, its presence in any of the organs, even in the smallest quantities, has to be accounted for; it is, therefore, very necessary that the toxicologist should carefully test all chemicals and re-agents employed in the detection of arsenic for traces of that substance. Its removal from organic admixtures, such as from the viscera or from the contents of the stomach, is best effected by taking advantage of the fact that the chloride of arsenic is volatile, and can be separated from organic matter by the process of distillation. For this purpose from two to four ounces of the liver, or of any of the other viscera to be examined, are minced and then thoroughly dried in a water-oven; when dry, the substance is powdered and drenched with strong and pure hydrochloric acid, with which it is allowed to digest in the cold for twenty-four hours. The mixture is then placed in a distil-

lation flask, which is adapted to a Liebig's condenser (fig. 3); the flask is placed upon a sand-bath and the contents heated until nearly dry. The acid distillate which contains any chloride of arsenic is received into a flask containing a little distilled water. The residue in the flask is then drenched a second time with hydrochloric acid, and submitted to a similar process of distillation. In this way all the arsenic is obtained in the distillate, and can be tested for by one or more of the following tests.

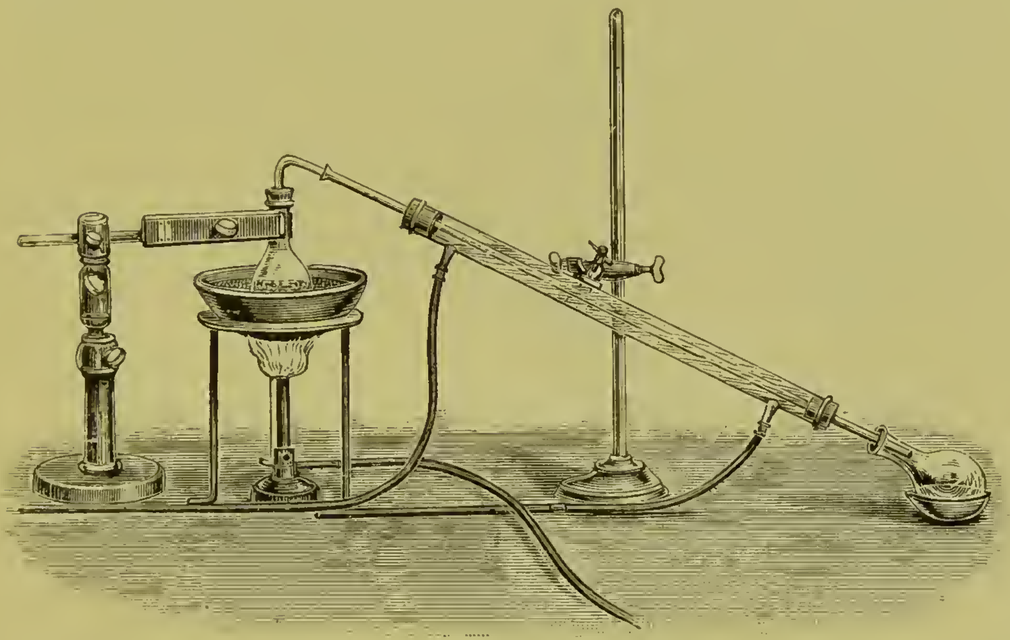


Fig. 3.—Apparatus for the Distillation of Arsenic

Of the various tests for arsenic, the most delicate, especially if the apparatus shown in fig. 6 be used, is Marsh's test.

Marsh's test.—This delicate test for arsenic depends upon the conversion of the arsenic into arseniuretted hydrogen, and then obtaining from the latter a deposit of metallic arsenic. Hydrogen is generated in a suitable apparatus (fig. 4) from pure zinc and pure sulphuric acid diluted with water; the gas is dried by passing it through a calcium chloride tube (fig. 4, A), and after the escape of the air from the apparatus, the hydrogen

is ignited as it issues from the end of the tube bent at a right angle. If some solution of arsenious acid, or of the chloride of arsenic obtained by the distillation process just described, be now poured into the apparatus by means of the funnel (fig. 4, B), the nascent hydrogen combines with the arsenic and produces arseniuretted hydrogen, the presence of which is quickly indicated by the hydrogen flame changing to a pale livid colour. If a piece of cold white porcelain, such as an evaporating dish or a porcelain crucible lid (fig. 4, C), be pressed down upon the flame for a moment or two, the cold porcelain becomes coated

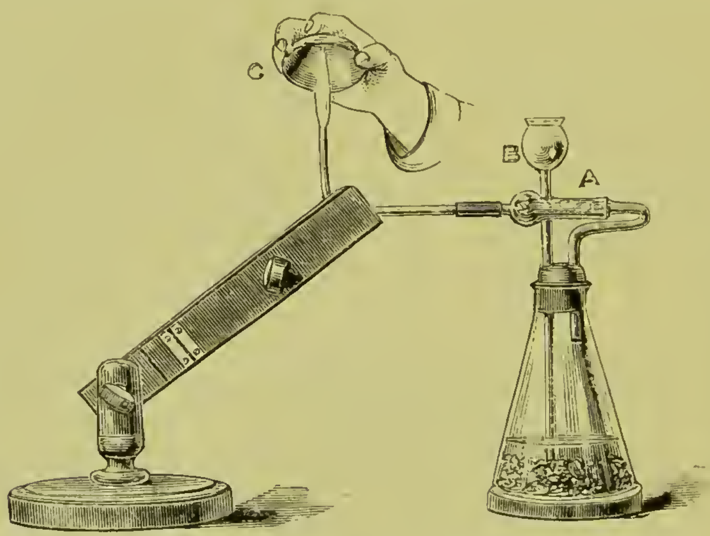


FIG. 4.—MARSH'S APPARATUS

with a blackish grey stain of metallic arsenic at the spot where the flame touches it. This is due to the fact that in arseniuretted hydrogen the two constituents are so loosely combined that the heat of the flame is sufficient to decompose the unburnt gas in the interior of the flame into metallic arsenic and hydrogen, and it is this finely-divided metallic arsenic which imparts to the flame the pale livid colour. Moreover, a low red heat is sufficient to decompose arseniuretted hydrogen into its elements, so that if, instead of testing the gas in the manner just described, it is passed through a glass tube, the central portion of which is maintained at a red heat (fig. 5), a mirror of metallic arsenic

(fig. 5, A) deposits on the cooler part of the tube just beyond the heated portion. As a somewhat similar stain is obtained in the detection of antimony by means of Marsh's test, the following methods may be adopted for the distinction of the arsenic and antimony stains. The deposit of metallic arsenic is soluble in a solution of bleaching-powder or of chlorinated soda, whereas the corresponding antimony deposit is insoluble. An even more delicate test is to place the stain obtained in Marsh's test in a porcelain capsule, heat it with a few drops of pure nitric acid, and then add a small quantity of a solution of ammonium molybdate in nitric acid. Antimony gives no

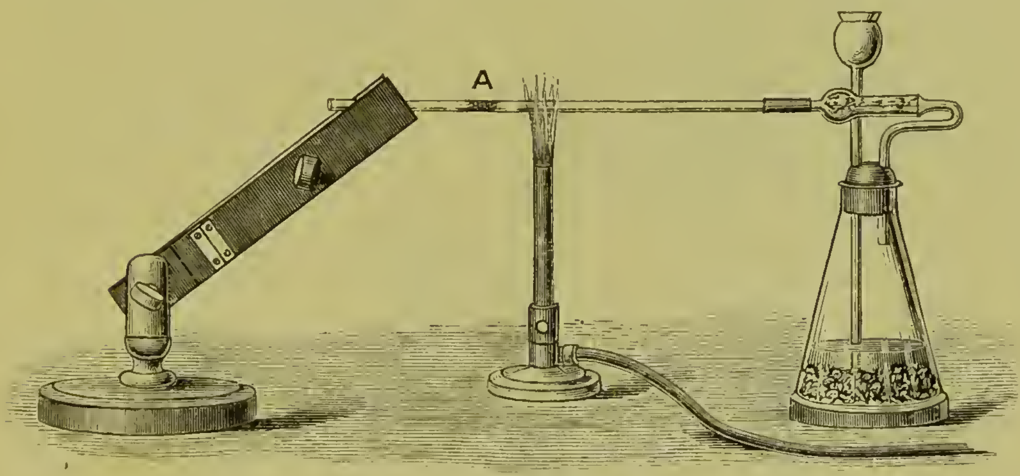


Fig. 5.—Marsh's Apparatus

precipitate, but if arsenic be present a yellow precipitate separates. The methods just described for the performance of Marsh's test are those usually employed in the demonstration of the test for lecture purposes, and also for the rough detection of arsenic; but for the detection of it in minute quantities the following arrangements constitute the most delicate means at the disposal of the toxicologist.

The apparatus (fig. 6) is a modification of that suggested by a Committee of the National Health Society.[1] Into the generating bottle (fig. 6, A), the stopper of which is ground to

[1] *Brit. Med. Jour.*, 1883.

the bottle so as to avoid the use of any kind of cork, pure zinc is introduced, and is covered with distilled water, and then strong sulphuric acid is added to generate hydrogen. For the delicate performance of this test it is not desirable that the evolution of the hydrogen should be too rapid; but, as the production of the gas from perfectly pure zinc and pure sulphuric

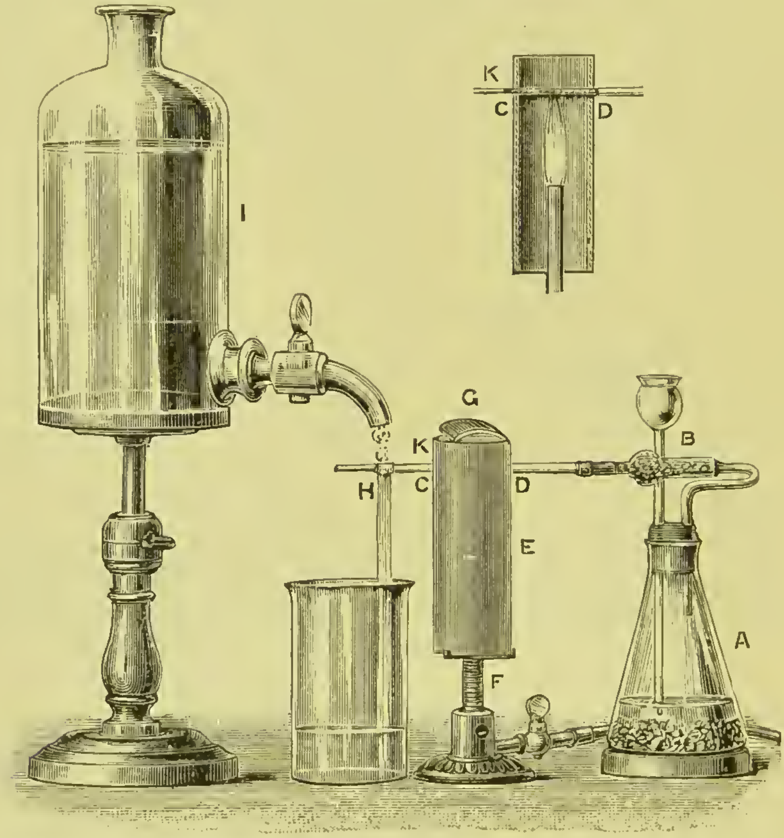


Fig. 6.—Apparatus for the delicate Detection of Arsenic by Marsh's Test

acid is sometimes very slow, its evolution may be facilitated by the addition of two or three drops of platinic chloride solution to the contents of the flask, previous to the addition of the sulphuric acid. The hydrogen is passed through a calcium chloride tube (fig. 6, B) to dry it, and is then made to pass through a tube of hard glass six inches long, the first portion of which has an internal diameter of one-quarter of an inch,

while the second portion, which has been sealed on to its end, has an internal diameter of one-eighth of an inch. At the junction of the two (fig. 6, c) a small plug of asbestos is placed, and the portion which traverses the chimney is filled with a mixture of dry sodium carbonate and charcoal, and at D another plug of asbestos is placed. The object of using sodium carbonate and charcoal mixture is to provide for the retention of any sulphur that may be present in the distillate from the viscera or contents of the stomach, which otherwise would interfere with the formation of the arsenical deposit. The hard glass tube traverses a fire-clay chimney (fig. 6, E), a section of which is shown; the chimney is one inch and three-quarters in diameter, and six inches high, and in the top edges two slots are filed to admit the tube to the depth of one inch. The tube is supported on a thin bridge of fire-clay of the same material as the chimney, the bridge being a quarter of an inch wide, one-eighth of an inch thick, and resting on the sides of the chimney at the bottom of the slots. The chimney surrounds a bunsen-burner (fig. 6, F) half an inch in diameter; on the top of the chimney is placed an arched clay cover (fig. 6, G). Round the narrower part of the glass tube, three-quarters of an inch from the chimney, is rolled a strip of calico (fig. 6, H), a quarter of an inch wide, which passes three or four times round the tube, and is then secured by a thread, the free portion hanging down into a beaker. Water is allowed to drop from a bottle (fig. 6, I) on to the portion of the calico wrapped round the tube at the rate of about 120 drops per minute. When the evolution of the hydrogen is steady, and all the air has been displaced from the apparatus (which may be ascertained by testing from time to time the explosive nature or not of the gas escaping from the further end of the glass tube), the escaping hydrogen and the bunsen-burner (fig. 6, F) are both lighted, and the portion of the glass tube traversing the fire-clay chimney is raised to, and maintained at, a red heat. The solution to be tested for arsenic is then poured down the funnel into the generating flask, small portions being added at a time,

and the action is allowed to continue for half an hour. If any arsenic be present, the formation of a brown or black mirror will be observed on the narrow part of the glass tube (fig. 6, K), between the clay chimney and the moist strip of calico; if no mirror be formed, then arsenic is absent from the solution that has been introduced into the apparatus. By this test it is possible to very readily detect $\frac{1}{5000}$ of a grain of arsenic, and even $\frac{1}{50000}$ of a grain produces a distinct stain. If required, a portion of the tube containing the deposit of metallic arsenic may be cut off and weighed, and if the arsenic is afterwards driven from it by heat, and the tube re-weighed, the difference in the two weighings will give the weight of metallic arsenic present; or the presence of arsenic may be verified by testing with ammonium molybdate dissolved in nitric acid (see p. 171); or the metallic arsenic may be converted into crystals of arsenious oxide by the process described in connection with Reinsch's test (see p. 175).

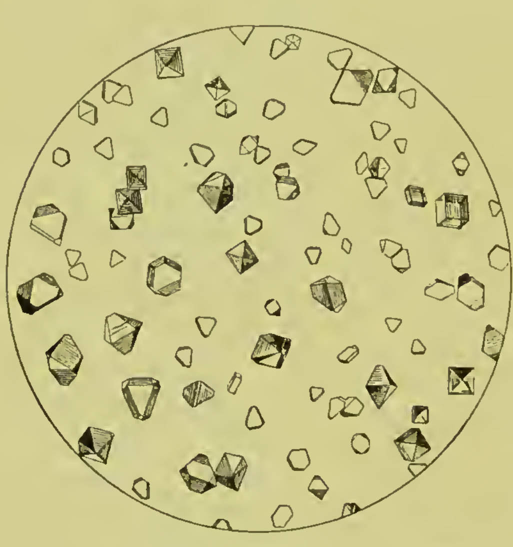


FIG. 7.—ARSENIC CRYSTALS OBTAINED BY REINSCH'S TEST
(Magnified 340 diameters)

Reinsch's test.—This test, if carefully performed, is also a very delicate one for the detection of arsenic. It consists in boiling, for a few minutes to half an hour, a strip of pure bright copper foil or roll of pure copper gauze in the solution to be tested, previously adding one-sixth its volume of pure hydrochloric acid. Under these conditions the metallic arsenic deposits on the surface of the copper as a dark steel-grey film, or if in minute quantities the film has a purplish colour. The acid liquid is then poured off, and the strip of copper is washed repeatedly with water, and dried, firstly, by pressure between folds of filter-

paper, and, lastly, by carefully warming it some distance above the bunsen-flame. The strip of copper with the deposit of arsenic on it is transferred to a perfectly clean and dry narrow test-tube, and heat is applied to the bottom of the tube where the strip of copper is lying, the tube being held in an almost horizontal position, with its mouth partially covered by the thumb of the operator. The metallic arsenic becomes volatilised, and then oxidises to arsenious oxide or white arsenic, which deposits on the cool part of the tube in small brilliant crystals consisting of perfect and imperfect octahedra. If the portion of the tube containing the sublimate is viewed under a microscope, using a quarter-inch or one-sixth-inch objective, the crystals will present the characteristic appearances shown in fig. 7. The following delicate modification of Reinsch's test is that recommended by a Committee of the National Health Society[1]; it consists in obtaining the deposit of metallic arsenic on a slip of copper by the method just described, and after carefully washing and drying the copper, it is cut into fine strips whilst held with a pair of forceps, the copper not being touched at any time by the fingers of the operator, as the presence of a trace of grease interferes with the subsequent delicacy of the test. A thin glass tube a quarter of an inch internal diameter and an inch and three-eighths long, sealed at one end and lipped like a test-tube at the other (fig. 8, A), is suspended by dropping it through a hole cut in a piece of stout sheet brass

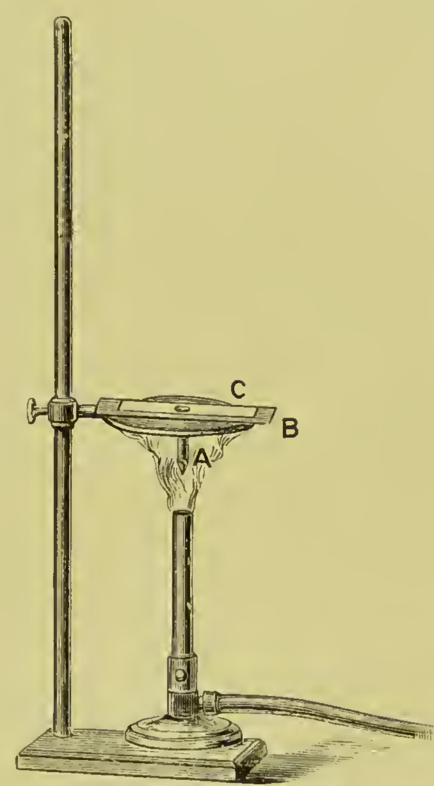


Fig. 8. — Apparatus for the delicate Detection of Arsenic by Reinsch's Test

[1] *Brit. Med. Jour.*, 1883.

four inches long by two wide (fig. 8, B), so that the lip just suspends the tube, and the brass plate is then placed on the ring of a retort-stand. The tube is heated nearly to redness to expel any traces of moisture, and when cold the copper strips are placed in it; above it, resting on the mouth of the tube, is laid a microscope slide (fig. 8, C), which has been previously warmed over a bunsen-flame till all the moisture first deposited has disappeared. The tube A is now heated with a bunsen-flame or spirit-lamp, the flame being allowed to play on the under surface of the brass plate; in a few seconds a sublimate appears on the slide, and the application of the heat should be continued until this sublimate begins to shrink from the edges, and forms a patch just the size of the bore of the tube; the flame should then be removed, the slide allowed to cool, and the sublimate examined under the microscope with one-sixth inch objective. If the sublimate be arsenic, it will be found to consist of octahedral and imperfect octahedral crystals (fig. 9). In the performance of this test it is important that the copper foil should be, previous to use, quite free from arsenic. The method of detecting that impurity in the copper is to boil together equal parts of a solution of perchloride of iron and strong hydrochloric acid, and, while boiling, introduce the slip of copper in a polished condition; if arsenic be present it is indicated in a short time by a black deposit on the copper, whereas if the metal be pure, its red colour will become more strongly marked. In addition to arsenic—antimony, mercury, bismuth, tin, silver, platinum, palladium, and gold are deposited

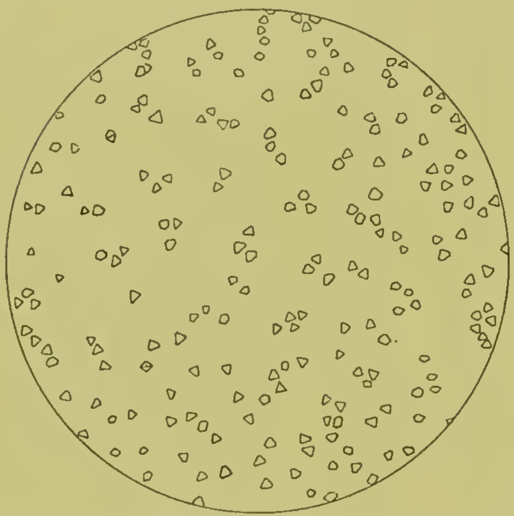


FIG. 9.—ARSENIC CRYSTALS OBTAINED BY REINSCH'S TEST FROM $\frac{1}{1000}$ OF A GRAIN OF ARSENIC. (Magnified 680 diameters)

on metallic copper when boiled with it in an acid solution, and if sulphur compounds are present in the solution, these may form a brown or black deposit of sulphide of copper on the surface of the foil. Of these deposits, three only—arsenic, antimony and mercury—yield sublimates on heating. The arsenical sublimate is crystalline, the antimony sublimate is amorphous (see p. 185), and the mercury sublimate when viewed under the microscope is seen to consist of globules of metallic mercury (see p. 193).

Wet tests for arsenic.—I. If to an aqueous solution of arsenic some copper sulphate be added, and a dilute solution of ammonia afterwards added drop by drop, a bright green precipitate of copper arsenite (Scheele's green) is thrown down. This precipitate is soluble in excess of ammonia, forming a dark blue solution.

II. If to an aqueous solution of arsenious acid some silver nitrate be added, and a weak solution of ammonia afterwards added drop by drop, a bright yellow precipitate of silver arsenite is obtained.

III. If stannous chloride, dissolved in strong hydrochloric acid, be added to a solution of arsenic in hydrochloric acid, a precipitate of metallic arsenic is thrown down. This test is a fairly delicate one.

IV. If sulphuretted hydrogen be passed through a solution of arsenic acidulated with hydrochloric acid, a yellow precipitate of arsenious sulphide is thrown down. This precipitate is soluble in alkalies and in ammonium sulphide. The verification of the arsenical nature of this yellow precipitate can be effected by drying and heating it with a mixture of three parts of dry sodium carbonate and one part of dried potassium cyanide, when a sublimate of metallic arsenic is obtained to which the various tests described on p. 171 may be applied. The distillate containing chloride of arsenic, which is obtained in the process of removing arsenic from the viscera (see p. 168), may be diluted with water and tested by the sulphuretted hydrogen test. The precipitate of sulphide of arsenic should be collected,

washed, dried, mixed with a mixture of dried sodium carbonate and potassium cyanide in the proportions just mentioned, and introduced into a hard glass tube similar to that described in connection with Marsh's test (fig. 10). Through this tube

Fig. 10.—Deposit of Metallic Arsenic in packed Exit-tube of Marsh's Apparatus

a stream of dried carbonic acid gas should be passed. On applying heat to the mixture of the arsenical sulphide and the sodium and potassium salts, a sublimate of metallic arsenic is obtained in the cool part of the tube anterior to the heated portion, in the position shown in fig. 10. This sublimate can then be submitted to appropriate tests.

Detection of arsenic in wall-paper.—I. A somewhat rough and ready test for the detection of arsenic in wall-paper is to place a square inch of the paper with the coloured surface uppermost in a small porcelain dish; distilled water is poured on so as just to cover the paper, and a few drops of solution of ammonia are added. After standing for a minute or two, a crystal of silver nitrate is dropped into the dish on to the surface of the piece of paper, when, if arsenic be present, a yellow deposit of silver arsenite will occur around the edge and over the surface of the crystal. The working of this test depends upon the solubility of Scheele's green (copper arsenite) in a weak solution of ammonia, and the precipitation from this solution of the yellow silver arsenite in the presence of excess of silver nitrate.

II. Reinsch's test may be employed for the detection of arsenic in wall-paper. Some of the paper is cut into small pieces, which are digested in dilute hydrochloric acid, with heat, for a few minutes. The acid liquid is then poured off and boiled with a strip of bright copper foil, and the test proceeded with in the manner described under Reinsch's test (see p. 174).

III. The best and most delicate test is to employ the modification of Marsh's test described on p. 172. The extract of the paper is made by taking five square inches of the wall-paper cut up in small pieces, and digesting them with warmth in either dilute hydrochloric acid or dilute sulphuric acid. This acid solution is then introduced into the generating bottle of the Marsh's apparatus, all the details described on pp. 171–174 being carefully observed. A paper, yielding, when tested under these conditions, a distinct black and lustrous deposit which is sufficient to cut off, at any point, a black line on a white ground, technically known as thick rule, should be condemned, provided the absolute purity from arsenic of the materials employed for testing has been determined. An estimation of the amount in the wall-paper may be made by comparing the mirror obtained with a set of standard mirrors obtained under similar conditions from known amounts of arsenic.

Estimation of arsenic: I. *By precipitation as arsenious sulphide with sulphuretted hydrogen.*—A measured quantity of the hydrochloric acid distillate obtained from the viscera by the process described on p. 168 is diluted with water, and a stream of pure sulphuretted hydrogen is passed through the liquid; the sulphuretted hydrogen is best prepared by applying heat to a solution of magnesium sulphydrate, when the gas is readily evolved in a pure state. After the complete precipitation of the arsenic as sulphide, a stream of carbon dioxide is passed through the liquid to displace the excess of sulphuretted hydrogen, and the yellow sulphide of arsenic is collected on a filter and weighed; or either of the two following methods may be employed for the weighing of the precipitated sulphide of arsenic in some other pure form.

(*a*) The precipitate of arsenious sulphide is dissolved in solution of ammonia, filtered if necessary, and the ammoniacal solution evaporated to dryness in a porcelain dish, and carefully heated on a sand-bath to carbonise any organic matter that may be present, without volatilising any of the arsenious sulphide. The residue is again dissolved in ammonia, filtered

if necessary, evaporated to dryness, and the residue dried in a water-oven and weighed as arsenious suphide.[1]

(*b*) Another method, advised by Bäckström,[2] consists in weighing the arsenic as pentoxide. The precipitate of arsenious sulphide is dried on the water-bath, and is oxidised by repeated careful additions of fuming nitric acid in small portions. The liquid is transferred to a platinum crucible, and, after evaporation, is heated to a temperature just short of redness to expel sulphuric acid. The residue, which is arsenic pentoxide, is then weighed rapidly, as it is a hygroscopic body.

II. A method described by Clark[3] is to obtain the arsenic in the form of a deposit on copper by Reinsch's process, by the repeated boiling of successive strips of copper foil with the substance to be tested, in the presence of dilute hydrochloric acid. When all the arsenic is thus deposited on the strips of copper, they are placed in a mixture of dilute caustic potash and peroxide of hydrogen and allowed to digest in the cold; the peroxide of hydrogen and alkali dissolve off the arsenic, forming potassium arsenate. The alkaline liquid is then poured off the copper, heated to boiling, filtered to get rid of oxide of copper, and evaporated to a small bulk. This is distilled with strong hydrochloric acid and ferrous chloride in a distillation flask, the distillation being repeated a second time. From the distillate, diluted with water, the arsenic is precipitated by means of sulphuretted hydrogen, and the precipitate is treated by one or other of the methods described above.

III. Another method of estimating arsenic consists in obtaining a deposit of metallic arsenic in the depositing tube of a Marsh's apparatus (see p. 171), and weighing, firstly, that part of the tube which contains the mirror, and, secondly, the same portion after the deposited arsenic has been dissolved off.

[1] Mohr, *Chem. Toxicologie*, 56. [2] *Zeit. anal. Chem.*, 31, 663-65.
[3] *Chem. Soc. Jour.*, 1893.

CHAPTER XII

Poisoning by antimony salts—Acute and chronic poisoning by antimony salts—Poisoning by mercury preparations - Acute mercurial poisoning—Poisonous preparations of mercury—Chronic mercurial poisoning—Poisoning by lead salts—Acute lead poisoning—Chronic lead poisoning —Industrial and accidental causes of lead poisoning.

Antimony

The preparations of antimony met with in toxicological enquiries are practically only two in number—viz. tartar emetic and chloride of antimony. Tartar emetic, or tartarated antimony as it is sometimes called, is an oxytartrate of antimony and potassium, and in small doses is used in medicine as a depressant expectorant. Tartar emetic has occasionally been sold by mistake for tartaric acid, and also for cream of tartar. Chloride of antimony, or *butter of antimony*, as it is commonly known, is an extremely acid liquid, containing the chloride of antimony dissolved in excess of strong hydrochloric acid. It is prepared by the action of strong hydrochloric acid on the native crude sulphide of antimony or black antimony, and, since that substance contains a quantity of iron, the butter of antimony of commerce is a liquid of a reddish-brown colour, from the presence of chloride of iron; it is technically known as *bronzing liquid.*

Symptoms.—The symptoms of poisoning by butter of antimony are those of antimony poisoning and, in addition, those of poisoning by hydrochloric acid, on account of the large amount of free acid contained in it, so that butter of antimony is not only a metallic poison, but a corrosive one as well. The symptoms that will now be described are those of poisoning by tartar

emetic. (i) A strong metallic taste, which comes on almost immediately after swallowing the poisonous dose. (ii) A feeling of great heat and constriction of the throat. (iii) A sensation of violent burning pain is generally felt in the stomach a few minutes after swallowing the poison. (iv) This is followed quickly by profuse vomiting. Blood may be present in the vomit, but is generally absent. (v) Profuse purging occurs, and is frequently accompanied by tenesmus. (vi) Faintness and extreme depression supervene. The pulse is small and rapid, the surface cold and frequently cyanosed, and bedewed with sweat. There is shivering, and the respiration is slow and laboured. (vii) Violent spasms of the muscles of the extremities sometimes occur. (viii) The urine may be almost entirely suppressed. (ix) Occasionally a comatose condition has followed the taking of tartar emetic, as in a case recorded by Dobie.[1]

Treatment and antidotes.—If vomiting has not occurred, it should be induced by means of an emetic of mustard and water, or by tickling the fauces with a feather, or the stomach-pump or tube may be employed if free vomiting does not occur. In cases of poisoning by 'butter or chloride of antimony' the stomach-pump should not be used. After the stomach has been emptied, a drachm of tannic acid, dissolved or suspended in warm water, may be administered, with the object of forming an insoluble tannate of antimony. Opium should be given to relieve the pain, and ice may be administered to allay the vomiting and gastric irritation. Demulcent drinks, such as linseed tea, almond mixture, and milk, may be taken, and warmth and stimulants should be employed, if necessary, to overcome the depressing action of the poison.

Post-mortem appearances.—In cases of acute poisoning by antimony the stomach and intestines are generally inflamed; ecchymoses of the mucous membrane are frequently present, and ulceration of the gastric mucous membrane is sometimes found. Inflammation may be absent in cases of chronic poisoning by tartar emetic. In the case of Mr. Bravo, who was poisoned

[1] *The Lancet*, 1887.

by tartar emetic in 1876, the stomach and duodenum were pale and yellowish on their mucous surfaces; there were ulcers in the cæcum, and the remainder of the large intestine was deeply blood-stained, but without ulceration. The lungs are frequently found congested in cases of poisoning by tartar emetic.

Fatal dose.—Probably from five to fifteen grains of tartar emetic represent a minimum fatal dose for a healthy adult, but recovery has taken place after taking 200 grains of this poison. It should, however, be remembered that a dose of tartar emetic, administered or taken at once, may not be attended with the same amount of danger to life as if the same quantity were given in small doses over a prolonged period of days, or even weeks. In two cases described by Taylor, three-quarters of a grain of tartar emetic proved fatal to a child, and two grains to an adult. In these cases there were diseased conditions present which favoured the fatal operation of the poison.

Fatal period.—The shortest time in which tartar emetic has produced a fatal result is seven hours, but death may occur after the lapse of several days. Small doses of tartar emetic may occasion the death of persons suffering from pneumonia and bronchitis, by the depressing influence of the drug on the heart and nervous system.

Chronic poisoning by antimony.—Tartar emetic has on several occasions been given with a criminal intent in small doses to invalids, and in the majority of cases of death from homicidal poisoning by tartar emetic this method of administration has been employed. In this way the nervous and circulatory systems become progressively depressed, and, on account of the persistent vomiting and purging, the retention of food is impossible, and so a fatal issue is brought about. The symptoms of chronic poisoning by antimony are similar in many respects to those previously detailed, but as they are apt to be mistaken for the symptoms of disease, a brief recapitulation of them may be advisable. They are:—Nausea, vomiting, chronic diarrhœa (sometimes alternating with constipation), small rapid pulse,

muscular weakness, cramps, depression, clammy skin, and cold sweats.

Analysis and tests: I. *Sulphuretted hydrogen test.*—Antimony sulphide is an orange-red precipitate, the colour being very characteristic. If hydrochloric acid be added to an aqueous solution of tartar emetic, a white precipitate of oxychloride of antimony is first thrown down, which, however, is soluble in excess of hydrochloric acid. If sulphuretted hydrogen be now passed through this acid solution, an orange-red precipitate of antimony sulphide falls. This precipitate is soluble in alkalies and in ammonium sulphide. To obtain this precipitation of antimony sulphide in the detection of antimony in the contents of the stomach or in the viscera, a tartaric acid extract should be obtained by heating the contents of the stomach, or the finely divided viscera, with a solution of tartaric acid on a water-bath for about half an hour; the matters should then be strained through fine cambric and the liquid concentrated by evaporation. Sulphuretted hydrogen gas should then be passed for some time through the liquid, when a dirty orange-red or brown precipitate will be thrown down, consisting of antimony sulphide, organic matter, and sulphur. This impure sulphide should be washed, transferred to a porcelain dish, and some pure nitric acid added; the whole should be evaporated to dryness, and, if necessary, the operation should be repeated until the organic matter is destroyed. The residue is then warmed with a solution of caustic potash, filtered, evaporated to dryness, and the residue fused; the cooled mass (which consists of potassium antimonate) is boiled with water acidulated with tartaric acid and filtered; from the filtrate sulphuretted hydrogen will throw down the characteristic pure orange-red antimony sulphide, or the filtrate may be employed for the detection of the antimony by Marsh's process.

II. *Reinsch's method.*—If to a solution of an antimony salt sufficient hydrochloric acid be added to dissolve any precipitate at first thrown down, and a strip of bright copper foil boiled in it, a purplish or dark grey film of metallic antimony, according

to the amount present, is deposited on the copper. If the strip of copper be then washed, dried, and heated in a small tube in the manner described in connection with Reinsch's test for arsenic, a white sublimate of antimony oxide is obtained, which differs from the arsenical sublimate in that it is amorphous, and is deposited on the sides of the tube much nearer the copper than the arsenical sublimate is, on account of the greater volatility of arsenic. If the sublimate be examined under the microscope, no trace of a crystalline appearance can be seen. The sublimate of antimony oxide thus obtained may be dissolved by warming it with a weak solution of tartaric acid, and if sulphuretted hydrogen be then passed through the solution, the orange-red coloured sulphide of antimony is thrown down.

III. *Marsh's test.*—The liquid to be tested for antimony may be examined by Marsh's process, as described in connection

FIG. 11.—DEPOSIT OF METALLIC ANTIMONY IN EXIT-TUBE OF MARSH'S APPARATUS

with the corresponding test for arsenic (see p. 169). If the deposit of metallic antimony be obtained upon a porcelain evaporating dish, it can be distinguished from the corresponding arsenical deposit by its being insoluble in a solution of chlorinated lime (bleaching powder), or of chlorinated soda, whereas the arsenic deposit is soluble in either of those solutions; or the stain may be heated with a few drops of pure nitric acid, and a small quantity of ammonium molybdate in nitric acid added, when antimony yields no precipitate, whereas arsenic produces a yellow one. If the deposit be obtained in the hard glass depositing-tube, it generally forms in two portions on either side of the heated part of the tube (fig. 11), the deposit on the side of the tube nearest to the generating bottle being much lighter than that on the opposite side; the two deposits are most widely separated at the under and hottest part of the tube. The deposit of metallic arsenic, obtained in a similar manner, only forms on

that portion of the tube furthest from the generating bottle (fig. 12). The reason for the different positions of the two deposits is, that metallic antimony is much less volatile than arsenic. At the edges where the deposit is fading away it does not show a brown appearance, such as is present in the case of arsenic.

Fig. 12.—Deposit of Metallic Arsenic in Exit-tube of Marsh's Apparatus

Antimony may be detected in the viscera by boiling a portion of the organs, finely cut up, in a mixture of one part of hydrochloric acid and five parts of water, the solution being tested by Reinsch's process, or by placing it in a platinum dish in which a small piece of pure tin or zinc is placed; where the metals come in contact with each other a black deposit of metallic antimony is formed either in a few minutes or in the course of a few hours. A more delicate method of detecting the antimony in the hydrochloric acid decoction of the viscus is to introduce into it a piece of platinum foil, around which a piece of zinc foil has been coiled, the acid decoction having been sufficiently diluted to prevent too violent an action on the zinc. After a time the platinum is coated with a deposit of metallic antimony, which may be washed off, nitric acid added to the residue, and the acid liquid evaporated to dryness; on dissolving the residue in hydrochloric acid, diluting, and passing sulphuretted hydrogen, the orange-red precipitate of antimony sulphide is thrown down.

Estimation of antimony.—From the hydrochloric acid decoction the antimony may be precipitated by means of sulphuretted hydrogen as sulphide of antimony, probably mixed with free sulphur; after the complete precipitation carbon dioxide is passed through the liquid to expel the excess of sulphuretted hydrogen. The precipitate is collected on a filter, washed, gently dried in a water oven, and transferred to a platinum boat, which is then introduced into a piece of

combustion tube, through which a stream of carbon dioxide is kept passing. The portion of the combustion tube upon which the platinum boat is resting (fig. 13) is then carefully heated,

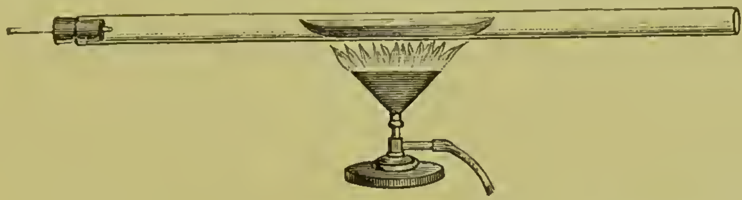


Fig. 13.—Platinum Boat in Tube for drying Antimony Sulphide Precipitate

until the orange-coloured sulphide is converted into the black sulphide; in this way the free sulphur and moisture are got rid of, and the residue, which consists of pure antimony sulphide in the black condition, can be weighed.

Mercury

Metallic mercury, in its ordinary condition, can scarcely be regarded as a poison, as only under very exceptional conditions has it ever produced toxic symptoms; but if applied to the skin or mucous membranes in a finely divided state, as in mercurial ointment or blue pill, or if it be introduced into the lungs in a state of vapour, then its toxic effects may be readily produced. The principal poisonous salt is the perchloride of mercury, or *corrosive sublimate*, also known as bichloride of mercury and mercuric chloride; this salt is soluble in water, but is much more so in alcoholic liquids. The other poisonous mercurial preparations are mercuric nitrate (present in the B.P. acid solution of nitrate of mercury), mercuric oxide or red precipitate, ammoniated mercury or white precipitate, and calomel, if taken in large doses or by persons peculiarly susceptible to its influence. Grey powder (mercury and chalk), if kept for a long time and exposed to light, may acquire toxic properties from a portion of the finely divided mercury becoming converted into an oxide.

Mercurial poisoning may be either acute or chronic.

Acute Mercurial Poisoning

Symptoms.—These come on immediately after or within a few minutes of swallowing a poisonous dose of mercuric chloride or corrosive sublimate. They are :—(i) A strong metallic taste. (ii) A feeling of constriction, with burning heat in the throat, passing down to the stomach. (iii) A violent pain in the abdomen, which pain is increased by pressure. (iv) Vomiting of white slimy masses, which are frequently streaked with blood. (v) Purging accompanied by tenesmus. Blood is frequently present in the stools. (vi) Collapse accompanied by a cold and clammy skin, cyanosis, and a small irregular pulse. (vii) Suppression of urine for twenty-four hours or longer not uncommonly occurs, and any urine that is passed generally contains albumen, and sometimes blood.

Acute mercurial poisoning may result from the external application of some mercurial preparation, or from the injection into the uterus, or into large abscess cavities, of a solution of corrosive sublimate. For instance, symptoms of acute mercurial poisoning, followed in some cases by death, have resulted from the external application of corrosive sublimate to tumours or ulcers, a method sometimes resorted to by quacks for the destruction of so-called cancer; similar toxic symptoms have resulted from injecting a solution of corrosive sublimate into psoas abscess cavities, and into the uterus after childbirth. A case is described by Sackur[1] in which a girl, aged twenty, sprained her wrist, and on the subsequent supervention of lymphangitis, mercurial ointment was applied and rubbed into some cracks on the hand. She subsequently suffered from several of the symptoms of acute mercurial poisoning, and five days after the inunction of the ointment, paralysis of the extremities supervened and the patient died; the amount of ointment used was small, and, in all probability, some idiosyncrasy must have been present to account for the ready and fatal toxic effect.

[1] *Berl. Klin. Woch.*, 1892.

Treatment and antidotes.—Some form of albumen should be administered at once in order to counteract the irritant effect of the corrosive sublimate, which is then converted into an insoluble albuminate of mercury; as quickly as possible after this the stomach-pump or tube should be employed to wash out the stomach, or an emetic should be administered. The albumen may be administered in the form of raw white of egg stirred up with water, or a tumblerful of milk may be given, or the vegetable albumen or gluten present in flour may be utilised, a tablespoonful of flour being administered in a tumblerful of milk or water. The albuminate of mercury so formed must, however, be removed by emptying the stomach by means of the pump, or by an emetic, as it may become ultimately digested and absorbed, or the mercury may be carried into the circulation owing to the solubility of the albuminate of mercury in excess of albumen. Opium should be administered to relieve the pain and purging, and opium enemeta employed if the tenesmus be distressing. Demulcent drinks should be freely given.

Post-mortem appearances.—The mucous membrane of the lips, mouth, and tongue is more or less whitened, and usually swollen and soft; a similar condition may be found in the mucous membrane of the œsophagus. The mucous membrane of the stomach is swollen, inflamed, and occasionally corroded. The small and large intestines are also sometimes found inflamed, but the large intestines are generally more affected than the small, the mucous membrane of the colon and rectum being frequently deeply injected, and presenting indications of hæmorrhage, and sometimes even of ulceration. If the poisoning be acute, parenchymatous inflammation of the kidneys may be present; but when the poisoning is more chronic, that form of inflammation is less marked, and interstitial nephritis is the more prominent condition. In the lungs Kaufmann has noticed thromboses in the capillaries, due, as he believes, to alteration of the blood.

Fatal dose.—The smallest quantity of corrosive sublimate

that has proved fatal is two grains, which occurred in the case of a child (age not given). From three to five grains of corrosive sublimate probably represent the minimum fatal dose for an adult, but recovery has taken place after swallowing 100 grains.

White precipitate, or ammoniated mercury, is not so poisonous as corrosive sublimate, but thirty-five grains of it have caused the death of an adult, although 120 grains, and even more, have been taken without a fatal result, when the treatment has been fairly prompt. Experiments made by Pavy on rabbits indicate that the toxic effects of this substance are more powerful than was formerly considered to be the case. Rabbits (which do not vomit) were killed by doses of four or five grains in a few hours, and after death mercury was found deposited in the various organs. The salt has been regarded either as non-poisonous or as owing its poisonous properties to variable amounts of perchloride of mercury contained in it; but this most probably is an incorrect view, the salt itself being doubtless a poisonous one.

Red precipitate, or mercuric oxide, in large doses produces symptoms of acute mercurial poisoning. In a case related by Ord,[1] in which a teaspoonful was taken, vomiting, diarrhœa and abdominal tenderness occurred; recovery took place.

Mercuric nitrate is a powerful corrosive owing to its being mixed with free nitric acid. Death has resulted from its internal use; half an ounce of the solution has caused the death of an adult; death has also resulted from the injection of it into the pregnant uterus, done with the object of procuring a criminal abortion, and also from its external use as an escharotic.

Calomel, or subchloride of mercury, or mercurous chloride, although commonly administered and regarded as a mild medicine, may occasionally destroy life even in comparatively small doses, owing to idiosyncrasy to the action of the drug; for instance, death has resulted from the administration of six grains of calomel to a boy aged fourteen.

[1] *The Lancet*, 1888.

Cinnabar, or vermilion (mercuric sulphide), is sometimes employed as the colouring matter in the vulcanised rubber used for mounting artificial teeth, and occasionally has been the cause of the following symptoms of mercurial poisoning, viz.—metallic taste in the mouth, inflammation or ulceration of the gums, weakness and want of nervous power, and eruptions on the skin.

Mercuric cyanide is an active poison, and is almost as powerful in its action as corrosive sublimate.

Turpeth mineral (mercuric oxysulphate) is also a poisonous mercurial preparation ; forty grains have proved fatal.

Mercury is eliminated in the urine, fæces, saliva, and sweat ; in the case of a mother suckling her child, it may be present in the milk.

Fatal period.—The shortest time in which corrosive sublimate has produced a fatal result is half an hour. Commonly from one to five days elapse in cases of acute mercurial poisoning ; in one case an adult took seventy grains of corrosive sublimate, and death did not occur till the twelfth day.

Chronic Mercurial Poisoning

The symptoms of chronic mercurial poisoning due to the taking of repeated small doses of mercury, or of one of its preparations, into the system are different to those of acute mercurial poisoning. They especially occur among workpeople engaged either in the preparation of the metal or in its employment for the preparation of various articles, such as amongst workers in quicksilver mines, looking-glass makers, thermometer and barometer makers, and also amongst those employing mercurial preparations, such as bronzers and furriers. Chronic mercurial poisoning is also occasionally produced by the application to the skin of a solution of some mercurial salt. A so-called 'skin tonic,' containing 1·6 grains of corrosive sublimate in each fluid ounce, has for some time been indiscriminately sold in this country as an application to the

skin for the purpose of improving the complexion; this highly injurious preparation had in one case produced somewhat severe mercurial poisoning, and the case in question came before one of the Dublin Law Courts. Formerly, when mercury in the form of blue-pill was given in large and repeated doses for the treatment of secondary syphilis, it was not an uncommon event for some of the symptoms enumerated below to be produced. The following are the symptoms of chronic mercurial poisoning, and the usual, though not universal, order in which they occur: (i) Nausea, anorexia, colic, and vomiting. (ii) General depression of the system, with loss of flesh and failure of strength. (iii) The salivary glands become inflamed and painful. (iv) Stomatitis, the gums and tongue becoming red, swollen, and sore, and the breath fetid. Acute periostitis of the jaw occasionally occurs, as in a case reported by Staveley and Pedley,[1] in which this affection resulted from the use of mercurial lotion for improving the complexion. (v) Salivation is a common symptom, and sometimes a blue line forms near the junction of the gums with the teeth. (vi) A peculiar cachexia, or form of anæmia, frequently supervenes. Erythematous, eczematous, or pustular rashes may occur. (vii) There may be mental excitability, passing into the mercurial tremors or trembles. These tremors commence in the tongue and face, then spread to the arms, and later to the legs; in character they somewhat resemble the tremors of paralysis agitans, in that the movements are fine; at first they are produced only by exertion, as in disseminated sclerosis, but subsequently the tremors become continuous; as a rule, they cease during sleep.

Analysis and tests.—I. Sulphuretted hydrogen gives with a solution of corrosive sublimate, acidified with hydrochloric acid, a black precipitate of mercuric sulphide. If the solution of the mercuric salt be strong, the precipitate generally at first comes down of a yellowish-white colour; then, as more sulphuretted hydrogen is passed in, the light-coloured precipitate passes

[1] *Brit. Med. Jour.*, 1893.

through successive stages of dark yellow, orange and brown, to a black colour. These colour-stages are due to the partial displacement, at first, of the acid radical of the mercury salt by the sulphur.

II. Potassium iodide gives with a solution of a mercuric salt a precipitate of red mercuric iodide; the precipitate is thrown down at first of a yellowish colour, which rapidly changes to red; it is soluble in excess of either the mercuric salt or of potassium iodide, but especially so in the latter.

III. If a solution of a mercuric salt be acidified with a few drops of hydrochloric acid, and then boiled for a few minutes with a strip of bright copper foil, metallic mercury is deposited on the surface of the strip of copper, imparting to it a characteristic lustre resembling polished silver. If the strip of copper be washed with water, carefully dried, and then heated in a small dry test-tube or reduction-tube, a sublimate of metallic mercury is obtained on the sides of the tube. This sublimate, when rubbed with a glass rod, produces characteristic globules of mercury, or, previous to such rubbing, the sublimate may be viewed under a one-inch power of the microscope, when the characteristic appearance of the globules of mercury is well seen (fig. 14). After examining the mercurial deposit under the microscope, the presence of the mercury can be verified by removing the piece of copper foil and placing a scale of iodine at the bottom of the tube, which is then corked up and left for a few hours; the vapour of the iodine unites with the mercury, and converts the mercurial film first into the yellow iodide,

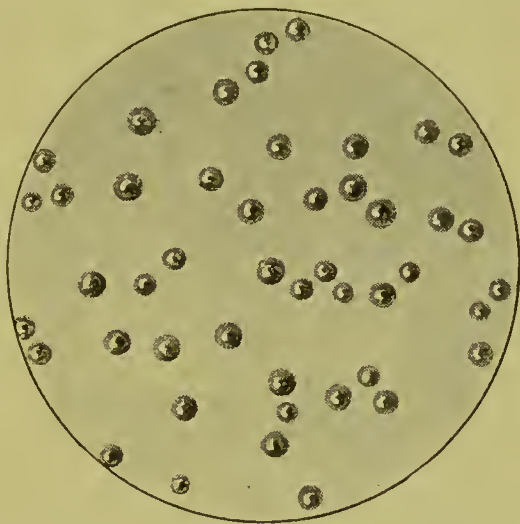


FIG. 14.—SUBLIMATE OF MERCURY GLOBULES
(Magnified 80 diameters)

which in its turn gradually changes to the scarlet mercuric iodide; this test is an extremely delicate one.

Instead of depositing the mercury on copper, the following delicate method for the detection of mercury may be employed. A small electrolytic couple is made by twisting a strip of gold foil round a piece of zinc foil; the solution to be tested is slightly acidulated with hydrochloric acid, warmed, and the electrolytic couple suspended in it for some hours; if mercury be present, even in very minute amount, the gold loses its colour, and acquires a silvery appearance from the deposition of metallic mercury on it, the zinc becoming more or less dissolved. The slip of gold foil is then washed, first in water, then in alcohol, finally in ether, and is dried by exposure to the air; if it be then submitted to heat in a small dry test-tube or reduction-tube, a sublimate of metallic mercury is obtained, which may be examined by the methods and tests previously described.

IV. Caustic potash and caustic soda both give with solutions of mercuric salts a yellow precipitate of mercuric oxide.

All the tests above described may be applied to white precipitate (ammoniated mercury) and to red precipitate (mercuric oxide) by previously dissolving these substances in hydrochloric acid of suitable strength.

Tests for mercurous salts.—I. Hydrochloric acid gives with a solution of a mercurous salt a white precipitate of mercurous chloride (calomel); this precipitate is blackened by the addition of ammonia.

II. Potassium iodide gives a dirty green precipitate of mercurous iodide with mercurous salts.

III. Caustic potash and caustic soda both give a black precipitate of mercurous oxide with mercurous salts.

Calomel can be detected by either of the two last-mentioned tests.

Distribution of mercury in the body.—Mercury is deposited in the kidneys, liver, spleen, and intestines; the large intestines always contain more than the small; traces of mercury are also found in the brain, lungs, and salivary glands; but, according to Ullmann, the saliva does not contain mercury, and, according

to that authority, salivation as the result of mercurialisation is to be regarded as a reflex phenomenon.

Estimation of mercury in the urine and viscera.—The urine is concentrated by evaporation and mixed with hydrochloric acid, or the viscus to be examined is minced, and then digested with equal parts of water and hydrochloric acid, and the organic matter present in the urine or organ to be examined is destroyed by the addition of potassium chlorate (see p. 117); the mercury is then removed from the solution by means of electrolysis. To effect this, the liquid, filtered after the destruction of the organic matter by hydrochloric acid and potassium chlorate, and subsequent heating to expel chlorine, is placed in a glass cell,[1] the bottom of which consists of a sheet of vegetable parchment. The cell is then immersed in an outer cell, containing distilled water acidulated with a few drops of sulphuric acid, so that the liquids in the inner and outer cells stand at the same level. A piece of gold foil is submerged in the liquid contained in the inner cell, and is connected with the cathode of four Grove's cells, while a piece of platinum foil connected with the anode is immersed in the outer cell; the pieces of foil are so placed as to be opposite to each other, but separated by the parchment diaphragm. The circuit is then closed for six or eight hours, after which the piece of gold foil is removed with the mercury deposit on it. It is washed first with water, then with alcohol, lastly with ether, and then carefully dried and weighed. The foil with the deposit is then introduced into a piece of hard glass tubing through which a current of dry air is passed, and sufficient heat is applied to drive off the mercury from the foil on to the tube; the gold foil is then re-weighed, the loss of weight representing the mercury previously deposited on it, and for control purposes the tube is weighed with the deposit, and again after it has been driven off by heat. Mercury may be estimated in saliva, if it be present in the secretion, by a similar process, but omitting the previous employment of potassium chlorate.

[1] Dixon Mann's *Forensic Med. and Toxicol.*, 1893.

Lead

The two salts of lead which are of particular medico-legal importance, are the acetate of lead or *sugar of lead*, and the carbonate of lead or *white lead*. In addition, toxic effects may be produced by the oxyacetate of lead as present in *Goulard's lotion*, by the red oxide of lead, and by chromate of lead; also, if fine particles of metallic lead are repeatedly taken into the system, poisonous effects are produced. Of the various lead compounds, the acetate is most frequently taken as a poison. It has a sweetish taste at first, and hence is called *sugar of lead*, but this sweetish taste is soon succeeded by an astringent metallic taste; it is not a very active poison, though it is commonly considered to be a virulent substance. The lead salts act as mild irritants. Accordingly as large doses are taken at once, or small doses at repeated intervals, the symptoms of acute or chronic lead poisoning result.

Acute Lead Poisoning

Symptoms.—(i) A short time after swallowing, a burning sensation is felt in the throat, with dryness and thirst. (ii) Vomiting generally occurs within about half an hour. (iii) Severe colic, the pain being relieved by pressure; the abdominal walls are tense and sometimes retracted. (iv) Constipation occurs; if any fæces are passed, they are of a dark colour from the presence of sulphide of lead. (v) The urine is frequently partially suppressed. (vi) Nervous symptoms may appear, consisting of prostration, pains in the head and limbs, cramps in the calves of the legs, general numbness, and occasional paralysis of the limbs.

Treatment and antidotes.—The stomach should be emptied and washed out by means of the stomach-pump or tube. Half an ounce of magnesium sulphate should be afterwards administered with the object of converting the lead salt present in the intestines into the insoluble sulphate of lead, and with

the further object of expelling the lead salt by producing profuse watery evacuations. Demulcent drinks should be given as long as symptoms of irritation remain, and opium, if necessary, to relieve the colic and severe vomiting that are generally present.

Post-mortem appearances.—Cases of lead poisoning are rarely fatal, and consequently but few opportunities have occurred of ascertaining the post-mortem appearances of poisoning by lead salts. The stomach and intestines have been found inflamed, and the mucous membrane partially eroded.

Fatal dose.—Nothing certain is known; probably the fatal dose of lead acetate for an adult is from one to two ounces. Recovery has taken place after swallowing one ounce of the acetate.

Fatal period.—Nothing certain is known.

Chronic Lead Poisoning

Chronic lead poisoning is extremely common. It may be produced either from industrial occupations, in which lead or some of its compounds are employed, or from accidental causes, the lead being introduced into the system in some form or other.

I. **Industrial causes of chronic lead poisoning.**—(*a*) Workers in white lead, red lead and other lead salts, such as painters, fitters, plumbers, workers at electric light works, where in the accumulator-works a paste of red lead and sulphuric acid is rubbed on the leaden plates; japanners; 'dippers' engaged in glazing earthenware, who have to dip the ware into a glaze consisting of borax, china stone, and red or white lead; workers engaged in the enamelling of iron plates, but especially those employed in the removal of the lead enamel to prepare the plate for advertising purposes; girls employed in combing and folding yarn dyed with the yellow chromate of lead (chrome yellow), from inhalation of the fine yellow dust given off during the manipulations. (*b*) Workers in metallic lead, such as

smelters; file-cutters, in connection with whose work the files are bedded in lead while being 'nicked,' so that much lead dust is produced and great leading of the hands and clothes occurs; compositors; workers engaged in fixing thin leaden caps on the tops of bottles containing sauces, &c.

II. **Accidental causes of chronic lead poisoning.**—These may be due to the accidental presence of lead in fluids, foods, or in any substance repeatedly brought in contact with the skin. The following are the principal sources from which lead may so gain access to the system:

(*a*) Drinking water contaminated with lead (see p. 199).

(*b*) Soda and seltzer water, drawn from siphons fitted with pewter or lead valves, have been found contaminated with small quantities of lead.

(*c*) Liquids containing free acid, such as vinegar and lemon-juice, may dissolve lead from glazed vessels, or from common tinned utensils, the tinning mixture of which may contain 60 per cent. of lead to 40 per cent. of tin. From such a coating, water alone will dissolve a fairly large quantity of lead. Wightwick [1] records three cases of chronic lead poisoning from eating tinned tomatoes; both lead and tin salts were found in the tomatoes and the juice. Home-made wine and beer brewed in glazed earthenware pans are very liable to be contaminated with lead.

(*d*) Farinaceous foods, chocolate, and tea if wrapped in lead foil, which frequently consists of lead thinly tinned, may become contaminated.

(*e*) Confectionary and buns are occasionally coloured with lead chromate (chrome yellow), and have produced toxic symptoms. In the colouring of buns this substance is used as a cheap substitute for eggs, and in order to give the desired rich yellow tint. According to Reese, a considerable number of cases of poisoning from this source have occurred in the United States within the past few years.

(*f*) Hair-dyes or so-called 'hair-restorers' may contain lead,

[1] *The Lancet*, 1888.

and then are liable to produce dangerous effects by their application to the head.

(*g*) Red lead may be employed as a cement for pipes or for cisterns, and so may become a source of contamination of drinking water.

(*h*) Cider or beer which has stood in lead pipes is very apt to dissolve lead, and acquire toxic properties. Chronic lead poisoning has not uncommonly occurred amongst those who have been in the habit of taking at public-houses the first draught of beer in the morning, which has necessarily been standing overnight in the pipes, and so especially becomes charged with lead.

(*i*) Lead shot are sometimes employed to clean out dirty wine bottles, and if some are accidentally left in the bottles, they may subsequently become acted on by the wine, and so contaminate it with lead salts.

(*j*) White lead and litharge are frequently used in connection with boiled oil to give leather a glossy finish. The glazed white leather lining of hats is sometimes so prepared, and the lead compound present may produce ill effects in a person wearing such a hat; either local discomfort may be produced, or symptoms of plumbism may occur from absorption of the lead salt after being acted on by the sweat.

Lead in drinking-water.—If lead be immersed in distilled water, which has been boiled to deprive it of all dissolved air, it remains unaffected by the water; but water containing air in solution attacks lead, the oxygen of the air forming lead oxide, which is then dissolved by the water as lead hydrate in small but appreciable quantities. This is one of the causes of the contamination with lead of a drinking-water which passes through leaden pipes, so that rain-water, when it is to be used for drinking purposes, should never be collected from a leaden roof, nor conducted through leaden pipes, nor kept in a cistern lined with lead. The corrosive action of water on lead is much increased by the presence in it of chlorides, nitrates, nitrites, and ammonia; whereas the presence of sulphates, and of

calcium carbonate held in solution as calcium bicarbonate by an excess of carbon dioxide, hinder the corrosive action ; in the latter case a film of lead carbonate is formed within the pipe, which is but very slightly soluble in water. The presence, however, of excess of carbon dioxide in the water increases its solvent action on the lead, the carbonate of lead being dissolved by water containing an excess of that gas, in the form of the more soluble lead bicarbonate. The presence of vegetable matter in water as a rule hinders the corrosion of lead, owing to the formation of an insoluble compound of lead oxide and organic matter. Some soft waters, notably some moorland waters, have a powerfully solvent action upon lead, due in some cases perhaps to the presence in the water of humic or ulmic acids derived from the soil, or perhaps, in other cases, to traces of sulphuric acid produced by oxidation of iron pyrites. Power has suggested that the action of water upon lead is dependent upon organisms. Crookes, Odling, and Tidy attribute the lead-dissolving power to the absence of silica from the water, and have shown that when dissolved silica is present in the water, lead becomes coated with a film of insoluble lead silicate. As an actual fact all drinking waters that have been allowed to flow through leaden pipes, or that have been stored in cisterns lined with lead, contain some lead in solution ; if, however, the amount of lead carbonate does not exceed $\frac{1}{60}$ of a grain per gallon, then its presence in drinking water may be neglected. The contamination of a water by lead may be due to the careless employment of white lead or red lead as a cement for junctions of pipes, or for cementing together a slate cistern.

It is not so much the quantity of lead taken that determines the symptoms of chronic lead poisoning as its continued introduction into the system. Moreover idiosyncrasy plays an important part in connection with lead poisoning, as only a small proportion of those exposed to the risk of chronic lead poisoning develop the toxic symptoms. Oliver[1] regards the abuse of alcohol as a most important factor in increasing the tendency

[1] *Lead Poisoning in its Acute and Chronic Forms*, 1892.

to chronic lead poisoning. Gouty subjects are more readily affected by lead, and lead also tends to develop gout. Oliver states that women are affected by lead more quickly and at an earlier age than men. Cases of chronic lead poisoning have been noted, in which symptoms apparently appeared years after the individual had ceased to be exposed to the action of the metal. In pregnant women chronic lead poisoning frequently leads to abortion.

Symptoms of chronic lead poisoning.—(i) Signs of impaired nutrition. (ii) The skin assumes a sallow or yellowish tint (the lead cachexia). (iii) A disagreeable astringent taste is felt in the mouth, and the breath is generally offensive. (iv) Pains in the stomach or abdomen, and constipation of the bowels occur. (v) A blue line is to be seen, in the majority of cases, near the edges of the gums, and generally better marked in the upper jaw. The line is due to the formation of lead sulphide, which is produced by sulphuretted hydrogen; the latter results from the decomposition of small particles of food collecting between the teeth and gums, and acts upon the albuminate or other compounds of lead present in the gums. Where the teeth are absent the blue line is also absent. (vi) Colic occurs, with obstinate constipation. The pain of the colic is as a rule referred to the region of the umbilicus, and is relieved by pressure. When the spasms of colic are present the abdominal walls are hard, frequently retracted, and the pain is not uncommonly accompanied by tenesmus. (vii) Arthralgia, or pains in the joints frequently occur. These pains are not uncommonly diagnosed, in cases of plumbism, as being due to rheumatism, and consequently the exciting cause of the pains may be entirely overlooked. They occur most frequently about the knees, less frequently about the elbows and shoulders. The seat of the pains is most probably in the sensory nerves of the muscles in the neighbourhood of the joints, and they are most likely due to neuritis. (viii) In a small number of cases of chronic lead poisoning paralysis occurs. The extensor muscles of the hands and fingers are most frequently attacked, the condition known

as 'wrist-drop' being the consequence; the supinator longus usually escapes. Both arms usually suffer, though one is generally more affected than the other. Occasionally the interossei of the hands are affected and waste. The other muscles more rarely affected are the deltoid, biceps, coraco-brachialis, and the extensors of the foot. These various paralyses are probably due to a peripheral neuritis. Lead is peculiar in its action upon the nervous system, in that it may attack both the central and peripheral portions. As the result of its action on the central nervous system, headache, vertigo, sleeplessness, excitability, hallucinations, or delirium may occur; or drowsiness may be produced, and not uncommonly convulsions attended with loss of consciousness. (ix) Ulceration of the mouth and of the inner sides of the cheeks has been noticed by Stedman [1] in children as a symptom of chronic lead poisoning.

Treatment of chronic lead poisoning.—The first part of the treatment must necessarily consist in the removal of the patient from the influence of the poison. Of the different remedies that have been employed with the object of promoting the elimination of the metal from the system, the one most commonly used is potassium iodide. The results, arrived at by a series of elaborate experiments made by Dixon Mann,[2] indicate, however, that potassium iodide possesses little, if any, value as an eliminator of lead from the system. The conclusions drawn by Mann are that lead is chiefly eliminated by the bowels, and to a much smaller extent by the kidneys; that when once the lead is deposited in the tissues, it exists as a stable compound upon which drugs exercise little or no power; that the best aids to the elimination of lead are baths, general massage, fresh air, good food, and other methods which improve the general health and promote healthy metabolism. On the other hand, although potassium iodide may not promote the elimination of lead, it is quite possible that it may in some other way be beneficial in cases of chronic lead poisoning. Symptoms that may require appropriate treatment in con-

[1] *The Lancet*, 1891. [2] *Brit. Med. Jour.* 1893.

nection with chronic plumbism are the colic, which is relieved by opium; the joint pains, which are relieved by hot fomentations, or by painting with iodine previous to the application of the hot fomentations; and the paralyses, which may require massage and electricity. Humphreys[1] recommends the employment of atropine, in full doses, in cases of lead poisoning; according to him, it relieves the colic and other pains, keeps the bowels open, and assists, directly or indirectly, in the removal of the lead.

Analysis and tests.—I. With a solution of a lead salt, sulphuretted hydrogen gives a black precipitate of lead sulphide.

II. Potassium iodide gives a bright yellow precipitate of lead iodide, soluble in boiling water, and crystallising in golden-coloured spangles as the solution cools.

III. Potassium chromate gives a yellow precipitate of lead chromate (chrome yellow), which changes to an orange-red colour (orange chrome) on boiling with caustic potash or caustic soda.

IV. Sulphuric acid gives a white precipitate of lead sulphate. This, however, is not a very delicate test for lead, as lead sulphate is slightly soluble in water.

Detection of lead in the viscera, urine, and fæces.—The viscus to be examined is dried in a water-oven, and incinerated in a porcelain dish. The ash is powdered, mixed with dilute nitric acid, and evaporated to dryness; the dry residue is then extracted with hot distilled water, and the filtered extract is tested either by the tests described above, or by the electrolytic method (see p. 195). For the detection of lead in the urine and fæces, Dixon Mann[2] recommends the evaporation of the urine to the consistence of gruel, and the mixing of the fæces with distilled water to a like consistence; the liquids are then treated with potassium chlorate and hydrochloric acid to destroy organic matter, and the filtrate after cooling is submitted to electrolysis, as described in connection with the detection of mercury (see p. 195), a piece of platinum foil being substituted for the gold foil connected with the cathode.

[1] *The Lancet*, 1891. [2] *Forensic Med. and Toxicol.*

CHAPTER XIII

Poisoning by copper salts—Zinc salts—Tin salts—Contamination of food by tin—Poisoning by chromium preparations—Silver nitrate—Iron salts—Bismuth salts.

Copper

The salts of copper commonly met with in toxicological enquiries are copper sulphate, known also as *blue vitriol* and *blue stone*, and the oxyacetate of copper or *verdigris*. Both these salts have been used for purposes of suicide, and for attempts at murder. In addition, copper sulphate has been frequently taken or administered for the purposes of procuring abortion; it is not an abortifacient, and any effect that it might possibly have in causing the expulsion of the contents of the uterus would be simply through its general action as an irritant to the gastro-intestinal tract, and so possibly indirectly affecting the uterus through the sympathetic nervous system.

Symptoms.—The symptoms of poisoning by a copper salt usually come on within five or ten minutes of taking it, and are: (i) A strong metallic astringent taste, with a feeling of constriction in the throat. (ii) Speedy and violent vomiting; the vomited matters at first are of a green or blue colour (a similar colour may occasionally be due to altered bile, which can be distinguished from the colour of a copper salt by the addition of excess of ammonia to the vomit. The colour, if due to bile, remains unaltered; if due to a copper salt, the blue colour is considerably intensified). (iii) Severe abdominal pain is felt, with thirst; purging occurs, and is frequently of a violent nature. (iv) Collapse, accompanied by, as a rule, pain in the head. (v) In aggravated cases spasms of the extremities, and

convulsions occur. (vi) The urine is generally diminished in amount.

Treatment and antidotes.—If vomiting occur, white of egg and warm water should be freely administered ; the white of egg is given to form the comparatively inert albuminate of copper, and the warm water to encourage the vomiting and removal of that compound. If vomiting do not occur, the contents of the stomach must be removed by means of the stomach-pump or tube, and the stomach thoroughly washed out. The subsequent treatment consists in the administration of bland mucilaginous drinks, and of morphine or opium, if necessary, to relieve pain and excessive vomiting.

Post-mortem appearances.—The stomach and intestines are inflamed, the mucous membrane may be softened and swollen, and the mucous membrane of the stomach may be eroded. If the copper salt has not been thoroughly removed by vomiting and purging, a blue or greenish colour of the mucous membrane of the stomach and intestines may be present.

Fatal dose.—Nothing definite is known of the fatal dose of copper sulphate ; half an ounce of verdigris has proved fatal in the case of an adult.

Fatal period.—The shortest time in which copper sulphate has killed is four hours, but usually the fatal result is delayed for several days.

Chronic Copper Poisoning

Chronic copper poisoning occasionally occurs from the employment of copper vessels for culinary purposes. Articles of food may be boiled in clean copper vessels without risk, provided the articles are free from acids ; but it is unsafe to keep articles of food or preserves in copper vessels in the cold, owing to the oxygen of the air acting on the metal at the point of contact of the contained substance with the interior of the vessel. It is preferable, if copper vessels are used for culinary purposes, or for the preparation of aerated waters, that they should be tin-lined ; it is not uncommon for traces of copper

to be found in soda-water, from the employment of condensers made of copper, which are either not lined with tin or improperly so. Chronic copper poisoning is also occasionally met with among workers in the metal and its salts, and also among brass-workers. Copper sulphate is sometimes used to give a rich green colour to preserved peas and other vegetables, olives, and green pickles; the employment of copper as an artificial colouring for articles of diet must be regarded as the admixture of a noxious substance, and therefore as rendering the article injurious to health. In France, however, the restrictions on the greening of preserved vegetables with copper have been recently removed. In green vegetables, the copper, to some extent, probably exists as an insoluble leguminate of copper, from which, however, the metal is liberated and rendered soluble by the action of the digestive juices. Tschirch [1] finds, as the result of some recent investigations as to the colouring of peas and other preserved vegetables with copper, that the production of the bright green colour is due to the formation of a copper salt of the phyllocyanic acid of the chlorophyll. Although this copper phyllocyanate is an insoluble compound, yet it has been found as detrimental as soluble copper salts. Tschirch, however, considers that a proportion of one part of copper in twenty thousand is unobjectionable, especially in view of the fact that in ordinary diet the daily quantity of copper taken into the system in the form of bread, meat, and vegetables, amounts to about one milligramme.

Symptoms of chronic poisoning.—(i) Loss of appetite, and symptoms of dyspepsia. (ii) Metallic taste in the mouth. (iii) Abdominal pain, generally of a colicky nature, with attacks of vomiting and diarrhœa. (iv) A green line on the margin of the gums has been noticed; by some observers the line has been described as indistinguishable from the blue line of lead. (v) Symptoms of peripheral neuritis, such as wrist-drop, cramps, &c., have been noticed in some cases.

[1] *The Pharm. Jour.*, 1894.

Copper is believed by some observers to be nearly always present in the liver of man. Dupré[1] found in human livers about one part of copper in 500,000; and in fourteen bodies, examined by Bergeron and Hote, copper in minute quantities was found in every case. It is also believed to occur normally in very small quantities in the kidneys and in the blood. The copper is probably mainly derived from the grains of cereals, such as wheat and barley; in turnips and most other vegetables, copper in very minute traces may generally be detected. It has been suggested by Johnstone[2] that the presence of copper in cereals may be due to the practice of dressing the grain, and also the ground, with sulphate of copper, with the object of protecting it from the ravages of vermin after the grain is sown.

Analysis and tests.—Copper may be detected in organic matter, such as in the viscera or urine, by drying and incinerating the organic matter in a platinum dish, using a bunsen burner provided with an iron tube (if a burner with the ordinary brass tube be used, copper is apt to be volatilised from the brass, and deposited on the incinerated residue); the residue is digested with nitric acid with heat, evaporated to dryness, and then extracted with a small quantity of water and filtered. To the filtrate the following tests may be applied.

I. Potassium ferrocyanide gives with solutions of copper salts a reddish-brown precipitate of copper ferrocyanide; this is a very delicate test for copper, as an extremely dilute solution of a copper salt produces a reddish-brown colouration on the addition of potassium ferrocyanide.

II. Solution of ammonia gives a light blue precipitate of copper hydrate, readily soluble in excess of ammonia, and producing a dark blue solution.

III. If a piece of bright steel, such as the polished steel blade of a knife or a polished needle, be immersed in a solution of a copper salt, it becomes, in a short time, coloured with a reddish deposit of the metal copper.

[1] *Analyst*, No. 13, 1877.

[2] *The Lancet*, 1890.

IV. Sulphuretted hydrogen passed into a copper solution gives a black precipitate of copper sulphide.

V. Minute traces of copper may be detected by immersing for some time a small couple of zinc foil with platinum wire coiled round it, in the solution to be tested, and then exposing the platinum wire to the vapour evolved by adding sulphuric acid to some crystals of potassium bromide; if a trace of copper has been deposited on the platinum wire, a deep violet colour is produced due to the formation of a compound of copper bromide and hydrobromic acid.

The quantitative determination of copper can be effected by placing a solution, acidified with hydrochloric acid, in a weighed platinum dish, and then connecting the dish with the cathode of a battery, a piece of platinum being suspended in the liquid and connected with the anode. In this way the copper is deposited as a metallic film on the inner surface of the platinum dish, and, when it is entirely so deposited, the dish may be washed, dried, and weighed; the increase in weight will then give the amount of copper present.

Zinc

Two salts are met with in connection with cases of poisoning by this metal—viz. the sulphate and chloride. Zinc sulphate, which is also known as *white vitriol*, is an irritant poison; it has been mistaken for magnesium sulphate or Epsom salts, and for oxalic acid. Zinc chloride is a much more powerful poison than the sulphate, as it is a corrosive as well as an irritant; it is present in Sir William Burnett's Disinfecting Fluid, which is a concentrated solution of zinc chloride in water, containing about 220 grains of the salt to the fluid ounce; this fluid has frequently caused death as a corrosive poison, when taken accidentally or suicidally. Zinc chloride is also present in soldering fluid, which is prepared by dissolving the metal zinc to saturation in strong hydrochloric acid.

Symptoms.—The symptoms of poisoning by zinc sulphate are not so severe as those of poisoning by zinc chloride.

The symptoms of poisoning by zinc sulphate are—(i) An astringent metallic taste. (ii) Pain in the stomach and abdomen. (iii) Violent vomiting, which comes on immediately after swallowing the salt. (iv) Severe purging.

Symptoms of poisoning by zinc chloride.—In addition to those above described, zinc chloride, on account of its corrosive action, produces a burning sensation in the mouth, pharynx, œsophagus, and stomach, which is generally felt immediately after swallowing the poison; the purging is accompanied by tenesmus, and blood is frequently present in the motions; as a rule, there is severe collapse. Subsequently, progressive anæmia and emaciation may result from the destructive effects of the poison upon the mucous membrane of the stomach and intestines, producing disorganisation of the lining membrane of the stomach, and consequent interference with the digestive processes; stricture of the œsophagus or pylorus may result, and so lead to wasting.

Treatment and antidotes.—The zinc salts produce such copious vomiting that the administration of an emetic, or the use of the stomach-pump, is unnecessary. White of egg mixed with milk should be freely administered, and carbonate of soda in dilute solutions may be given, with the object of converting the soluble zinc salt into the insoluble carbonate. The subsequent treatment consists in the administration of bland demulcent drinks, and of opium, if necessary, to relieve the pain, and to allay the purging.

Post-mortem appearances. In cases of poisoning by zinc sulphate the mucous membrane of the stomach and intestines is found inflamed. In cases of poisoning by zinc chloride much more severe effects are produced; indications of corrosion are found, as a rule, from the mouth, down the œsophagus into the stomach, and sometimes extending as far as the duodenum. The mucous membrane may be softened and white, or hardened and leathery, and in parts may be detached; in addition, it shows the usual signs of acute inflammation. In those cases in which the patients have survived for some weeks,

but have eventually died of emaciation, the mucous membrane of the stomach may be found either completely disorganised or replaced in parts by cicatricial tissue. In a case of poisoning by zinc chloride, described by Jalland,[1] in which death occurred eighty-seven days after taking about three or four ounces of a saturated solution of impure chloride of zinc with suicidal intent, absolute destruction of the stomach was found at the post-mortem examination ; the stomach was found to be represented by an organised inflammatory matting of the gastro-hepatic omentum, and the upper portion of the great omentum to the adjacent viscera, as follows : behind and below to the pancreas, the pillars of the diaphragm, and the vertebral column ; above, to the under surface of the left lobe of the liver ; on the left side, to the upper portion of the inner surface of the spleen.

Fatal dose.—Death has occurred in an adult after taking an ounce and a half of zinc sulphate. This is about the only case in which a definite quantity of this salt has been known to produce death. Recovery has followed the taking of one ounce of the salt. Zinc sulphate cannot be regarded as an active poison, since in cases of epilepsy it is frequently given as a nervine tonic, and, commencing with small doses, it has ultimately been given in forty-grain doses three times a day for a period of three weeks, without producing toxic symptoms. Zinc chloride, however, is a powerful poison, six grains having proved fatal, although recovery has taken place after taking two hundred grains. Anderson[2] describes a case in which a child, aged fifteen months, swallowed a teaspoonful of chloride of zinc soldering-fluid, and in which recovery took place after three days.

Fatal period.—In the case referred to above, in which a woman swallowed an ounce and a half of zinc sulphate, death ensued in thirteen and a half hours. The most rapidly fatal case of poisoning by zinc chloride that has been recorded is one in which death occurred in four hours. On the other hand,

[1] *Brit. Med. Jour.*, 1887. [2] *Ibid.*, 1893.

chloride of zinc may destroy life by producing stricture of the œsophagus or pylorus, or by leading to disorganisation of the mucous membrane of the stomach, and consequent gradual death from exhaustion and emaciation. In the case referred to above, as described by Jalland, death occurred eighty-seven days after taking chloride of zinc; and Tuckwell[1] describes a case in which death occurred from emaciation 116 days after taking three-quarters of a teacupful of 'Burnett's Disinfecting Fluid.' The fatal termination may even be prolonged considerably over this last-mentioned period.

Chronic zinc poisoning may result from the presence of zinc in drinking water, or from the preparation of food in zinc vessels, or amongst those engaged in the smelting of zinc. If water be conveyed through galvanised iron pipes, which are coated internally with zinc, or if it be stored in galvanised iron vessels, the dissolved oxygen and free carbonic acid gas of the water exert a solvent action upon the zinc; in addition, the solvent power of a water or of articles of food upon zinc is increased by the presence of chlorides.

Symptoms of chronic zinc poisoning.—These consist of interference with the processes of digestion, colicky pains, and either constipation or diarrhœa; in some cases symptoms of peripheral neuritis have been observed. Thresh[2] mentions the case of a child who suffered from marked constipation, from the contamination by zinc of water that had been conveyed through galvanised iron pipes.

Analysis and tests.—Zinc may be separated from organic admixture by digesting the organic matters with dilute acetic acid at a gentle heat, filtering, concentrating the filtrate, and then precipitating the zinc with sulphuretted hydrogen. The precipitated zinc sulphide (which generally contains organic matter), is collected on a filter, washed, and dissolved in strong nitric acid. The acid solution is evaporated to dryness, dissolved in distilled water, and subjected to the following tests:—

I. Ammonium sulphide gives with a neutral or alkaline

[1] *Brit. Med. Jour.*, 1874. [2] *Ibid.*, 1893.

zinc solution a white precipitate of zinc sulphide. This is a characteristic reaction, as zinc sulphide is the only white sulphide capable of being precipitated in such a manner.

II. Solution of ammonia gives a white precipitate of zinc hydrate, readily soluble in excess of ammonia. From this solution in the excess of ammonia the zinc may be precipitated as the white sulphide, by the addition of ammonium sulphide.

III. Potassium ferrocyanide gives a white gelatinous precipitate of zinc ferrocyanide. This test may be used to distinguish zinc sulphate from magnesium sulphate and oxalic acid, neither of which yields a precipitate with potassium ferrocyanide.

IV. Caustic potash and caustic soda both precipitate the white zinc hydrate, which is soluble in excess of either alkali, provided the latter be free from carbonate.

Tin

Poisoning by salts of tin is a very exceptional occurrence, and is then generally due to the use of tinned fruit, such as cherries, apples, pineapples, tomatoes, &c.—the acid juice of which acts upon either the solder of the tin, or possibly on the surface, which may have been coated with impure tin; in either way a soluble salt of tin may be formed and dissolved by the juice.

Symptoms.—(i) Nausea, occasionally accompanied by a metallic taste. (ii) Abdominal pain, followed by vomiting and diarrhœa. (iii) Symptoms of collapse may occur, with feeble, irregular, and rapid pulse, and occasionally cyanosis. (iv) If collapse be severe, unconsciousness may supervene. (v) Cramps in the legs sometimes occur, and the urine may contain a small quantity of albumen.

Treatment.—The stomach should be emptied either by the administration of an emetic of mustard and water, or by means of the stomach-pump or tube. Brandy should be given if there be much collapse; opium to relieve the pain, and demulcent drinks should afterwards be freely administered.

Post-mortem appearances.—Nothing is known, but probably if a fatal case of tin poisoning occurred the appearances of gastro-enteritis would be found.

Fatal dose.—Nothing is known. In four cases of tin poisoning, caused by tinned cherries, that were seen and described by the author and Metcalfe,[1] an estimation of the amount of tin present in the cherry-juice was made, as the tin from which the patients had taken the cherries was left half full of cherries and juice, and therefore was available for purposes of analysis. The juice was strongly acid, and the analysis showed that the acidity was mainly due to malic acid; the juice contained tin in solution equal to 1·9 grains of the higher oxide of tin in each fluid ounce, which would be equal to 3·2 grains of the malate of tin in the same quantity of juice. The symptoms of these cases were very severe, there being a considerable amount of collapse and cyanosis, and in one case unconsciousness; it was estimated that the symptoms were produced by doses of the malate of tin varying from four to ten grains. All the cases recovered.

A peculiar case is recorded[2] in which a chemist died from tin poisoning. He had inadvertently filled his pepper caster with putty powder, which is the higher oxide of tin, and had continued to use it until he fell ill a couple of months afterwards, and ultimately died.

Fatal period.—Nothing is known.

Analysis and tests.—Tin may be separated from organic admixture by destroying the organic matter by the moist method (see p. 117). The clear fluid may then be submitted to the following tests:—

I. Sulphuretted hydrogen gives with a solution of a stannous salt a brown precipitate of stannous sulphide, and with a solution of a stannic salt a yellow precipitate of stannic sulphide.

II. Stannous chloride added to a solution of mercuric chloride first reduces it to the mercurous state, throwing down a white precipitate of calomel, and subsequently reduces this

[1] *Brit. Med. Jour.*, 1890. [2] *The Med. Press and Circ.*, 1894.

to the metallic state, producing a grey precipitate, due to the formation of finely divided mercury.

III. With gold chloride stannous chloride gives a purple precipitate.

Chromium

The compounds of chromium that may come under the notice of the toxicologist are potassium bichromate, chromic acid, and lead chromate. Potassium bichromate, known also as *red chromate of potash*, and technically as 'red crystal,' is employed for staining furniture, and in some dyeing processes.

Symptoms.—The symptoms of poisoning by potassium bichromate are :—(i) An unpleasant taste, followed by pain in the stomach and abdomen. (ii) Vomiting, the vomited matters possessing at first a yellow colour, while later on the vomit may contain some blood. (iii) Diarrhœa occurs, the evacuations probably being of a greenish colour from the reduction of the bichromate to a green salt of chromium. (iv) Prostration and collapse, with pallor and a moist clammy surface; the pulse is small, feeble and irregular, and unconsciousness occasionally supervenes. (v) Respiration may become slow, and gasping, a quarter of a minute sometimes intervening between the respiratory acts. (vi) The pupils are generally dilated, and sometimes do not react to light. (vii) Cramps in the legs sometimes occur.

Chromic acid (CrO_3), or chromic anhydride, as it should properly be called, is used externally as a caustic and escharotic. A fatal case from the external use of it is recorded by White,[1] which resulted from a single application of about half an ounce of solution of chromic acid, containing one hundred grains to the ounce, to a mass of papillary growths on the external genitals; the patient died in a state of collapse twenty-seven hours after the application.

Treatment and antidotes.—The stomach should be washed out by means of the stomach-pump or tube. Opium should be

[1] *University Med. Mag.*, 1889.

employed to relieve the pain, and stimulants for the collapse. Demulcent drinks should be given freely in cases of poisoning by chromic acid. Magnesia or chalk may be given in milk with the object of neutralising the acid.

Post-mortem appearances.—The mucous membrane of the stomach is generally acutely inflamed, and more or less destroyed. It is either of a dark brown or greenish colour; if the latter, it is due to reduction of the bichromate to the green oxide of chromium. The upper part of the duodenum is generally found inflamed; the blood may be chocolate in colour.

Fatal dose.—Two drachms (120 grains) have proved fatal to an adult. Philipson [1] describes a case of recovery after taking 273 grains of potassium bichromate.

Fatal period.—The shortest fatal period in connection with potassium bichromate poisoning is forty minutes. This occurred in a case described by Stewart [2] in which a patient, a female aged forty-six, took about an ounce of the salt. In another case death resulted four hours after taking the poison.

Workers in bichromate factories frequently suffer from the bichromate dust that is drawn into the respiratory passages, and that also deposits on the surface of the skin. The dust, if inhaled through the nose, is especially apt to set up irritation of the nasal septum, and may lead to perforation and even necrosis of the septum. In addition, if there are any excoriations on the skin, cutaneous maladies may be set up from the irritant effects of the bichromate dust. Eruptions, somewhat similar to eczema or psoriasis, may be produced, and ulcers may form which in appearance somewhat resemble hard chancres. In connection with poisoning by lead chromate the effects are more due to the lead than to the chromium (see pp. 197, 198).

Tests.—I. Silver nitrate gives a bright red precipitate with potassium bichromate.

II. Lead nitrate gives a bright yellow precipitate with potassium bichromate.

[1] *The Lancet*, 1892.

[2] *Brit. Med. Jour.*, 1888.

III. If a dilute solution of potassium bichromate be acidified with hydrochloric acid, and sulphurous acid then added, a green colour is produced, due to the formation of chromium chloride. A similar effect is produced by substituting alcohol for sulphurous acid, and subsequently warming the mixture.

Silver

Silver nitrate in the solid state acts as a violent irritant and corrosive. Poisoning by silver nitrate is extremely rare, and, when it has occurred, has resulted from the accidental swallowing of a stick of nitrate of silver (lunar caustic) whilst being employed as an application to an ulcerated throat.

Symptoms.—(i) Pain in the œsophagus and stomach. (ii) Speedy vomiting; the vomited matters may contain blood. (iii) Purging generally occurs, and blood may be present in the motions. (iv) Collapse. (v) Cramps occasionally occur.

Treatment and antidotes.—Common salt dissolved in water should be given at once, with the object of forming the insoluble chloride of silver; this should then be removed either by the administration of an emetic of mustard and water, or by the use of the stomach-pump or tube, unless the salt itself produces copious vomiting.

Post-mortem appearances.—In a case of acute poisoning, the caustic effect of the poison may be indicated by streaks or patches of a greyish-white colour on the parts of the mucous membrane with which the silver salt comes in contact. The stomach shows the appearance of more or less general inflammation, which may also be found in the duodenum.

Fatal dose.—Fifty grains of nitrate of silver have proved fatal to an adult.

Fatal period.—The shortest fatal period that has been described occurred in connection with a child, aged fifteen months, who died six hours after swallowing a portion of a stick of lunar caustic.

Tests.—I. Hydrochloric acid gives a white curdy precipitate of silver chloride, insoluble in nitric acid, but soluble in solution of ammonia.

II. Potassium chromate gives a red precipitate of silver chromate.

III. Lime-water produces a brown precipitate of silver oxide.

IV. Sodium phosphate gives a yellow precipitate of silver phosphate.

Iron

The salts of iron that, in large doses, may act as poisons, are, ferrous sulphate, known also as *protosulphate of iron*, *green vitriol*, *copperas*, and *green copperas*, and ferric chloride, known also as *perchloride of iron*. The latter compound is contained in three preparations of the British Pharmacopœia, viz. a strong solution containing 254 grains of ferric chloride in each fluid ounce, and a weaker solution and a tincture, which are one-quarter the strength of the above-mentioned, and contain 63½ grains in a fluid ounce. Both ferric chloride and ferric sulphate, but especially the former, have been employed in poisonous doses for the purpose of attempting to procure abortion (see ii. p. 180). The tincture of perchloride of iron is commonly known under the name of 'steel drops.'

Symptoms.—Large doses of iron salts produce a metallic taste, pain in the stomach, vomiting and purging; the motions are black from the formation of sulphide of iron.

Treatment and antidotes.—An ounce of carbonate of soda dissolved in water, or stirred up with milk, should be given, and the stomach afterwards evacuated and washed out by means of the stomach-pump or tube. Demulcent drinks should then be freely given, and opium, if necessary, to relieve the pain and purging.

Post-mortem appearances.—Those due to gastro-enteritis. The mucous membrane of the stomach has been found thickened towards the pyloric end, and discolouration of the gastro-intestinal mucous membrane, from the formation of sulphide of iron, may be seen.

Fatal dose.—Christison relates a case in which an ounce

and a half of the weaker solution of perchloride of iron (the same strength as the tincture) caused death in a male adult in about five weeks. A case of recovery from an ounce of the tincture has been recorded.

Tests.—I. Potassium ferricyanide gives with a solution of ferrous sulphate a dark blue precipitate.

II. Potassium sulphocyanide gives with ferric chloride a bright red colour of ferric sulphocyanide.

III. Potassium ferrocyanide gives with ferrous sulphate a precipitate at first almost white, but rapidly changing to a light blue colour from the absorption of oxygen from the air. With ferric chloride potassium ferrocyanide gives a dark blue precipitate of prussian blue.

Bismuth

Very occasional cases of poisoning have resulted from large doses of the subnitrate of bismuth. This is of interest, since, though subnitrate of bismuth is an insoluble substance, it may act as an irritant poison if administered in large quantities. The irritant poisonous effects of the subnitrate of bismuth have been ascribed to the presence of arsenic as an adulteration, but toxic effects may be produced without such an adulterant. Subnitrate of bismuth is known also as *pearl white* and *magistery of bismuth.*

Symptoms.—(i) Metallic taste. (ii) Pain in the stomach. (iii) Vomiting and diarrhœa, the evacuations having a blackish colour from the formation of sulphide of bismuth. (iv) Collapse may occur. (v) Salivation, with inflammation of the gums, and offensive breath. The odour of the breath has been ascribed to the presence of tellurium occurring as an impurity in the bismuth salt.

Treatment.—Removal of the poison by washing out the stomach with the stomach-pump or tube, and the subsequent employment, if necessary, of opium and demulcent drinks.

Post-mortem appearances.—Those of gastro-enteritis.

Fatal dose.—Two drachms of subnitrate of bismuth have caused the death of an adult in nine days. Taylor quotes a case mentioned by Traill, in which a man took six drachms of the subnitrate in divided doses in three days. He suffered from vomiting, and pain in the abdomen and throat, but finally recovered.

Fatal period.—The shortest time known is that mentioned above, viz. nine days.

Tests.—I. If the subnitrate of bismuth be dissolved in strong nitric acid, and the solution poured into an excess of water, a white precipitate is produced.

II. Sulphuretted hydrogen gives with bismuth salts a black precipitate of bismuth sulphide.

ORGANIC POISONS

CHAPTER XIV

Poisoning by hydrocyanic acid—Potassium cyanide—Preparations containing hydrocyanic acid—Acute alcoholic poisoning—Distinction of different varieties of coma—Poisoning by chloral hydrate—Chloroform—Sulphonal—Nitroglycerine—Carbon bisulphide.

Hydrocyanic Acid

Hydrocyanic acid, or *prussic acid*, as it is commonly known, is a very powerful and rapidly-acting poison. In its pure state it is extremely poisonous, and is only occasionally prepared as a laboratory curiosity; in a dilute condition it is used in medicine in two forms—the Hydrocyanic Acid of the British Pharmacopœia, which contains two per cent. of the pure acid, and Scheele's Hydrocyanic Acid, which contains five per cent. of the pure acid. Hydrocyanic acid is feebly acid to litmus paper, and possesses a peculiar penetrating odour; if kept for some time with exposure to light, it slowly decomposes, and so loses strength, and, in addition, if the bottle containing it is not well stoppered, the strength of the acid rapidly diminishes by evaporation.

Symptoms.—With large doses the symptoms usually commence within a very few seconds of swallowing the poison, but they may be delayed for a period of thirty or forty seconds; it is quite possible for a person who has taken a poisonous dose of hydrocyanic acid to be able, before becoming insensible, to walk across a room or up a flight of stairs, and perform such actions as putting away the bottle or vessel from which the poison has been

taken. The symptoms are, however, very rarely delayed beyond one or two minutes. It is a poison which produces insensibility and loss of muscular power more quickly than any other. The symptoms are:—(i) Rapid loss of muscular power, giddiness, and the person quickly falls to the ground. (ii) Insensibility rapidly supervenes. (iii) The eyes are open, staring, and glistening; the pupils are dilated and do not react to light. (iv) The breathing is at first of a gasping nature, but soon becomes convulsive, with prolonged intervals between the breaths; the inspirations are usually very short and sudden, and the expirations very prolonged; the breathing may cease for some seconds, and the person appear to be dead, and then another convulsive breath may occur. If the insensibility be profound, the breathing is sometimes stertorous. This is important to remember, as, in connection with the death of Walter Palmer in 1856, it was contended that the fact of the deceased having had stertorous breathing was a proof that he died from apoplexy, and not from hydrocyanic poisoning. (v) The breath of the patient smells of hydrocyanic acid, and a similar odour is generally noticeable in the room. (vi) The pulse is feeble, rapid, and is frequently imperceptible at the wrist. (vii) Nervous symptoms soon supervene. In the early stage they consist of tonic spasms, which include spasmodic closure of the jaws, firm clenching of the hands, and convulsions of the trunk and limbs. These spasms are followed by complete relaxation of the muscles. (viii) The lips are frequently coloured with froth, and vomiting occasionally occurs. Involuntary micturition and defæcation also occasionally occur. Death usually takes place within five or ten minutes.

Death is in all probability due to arrest of respiration. According to Geppert the arrest of respiration is caused by the hydrocyanic acid diminishing or annulling the power of the tissues to take up oxygen, so that the oxygen accumulates in the blood. According to Kobert[1] the hydrocyanic acid forms with methæmoglobin a red compound, cyanomethæmoglobin,

[1] *Apoth. Zeit.*, 1891.

which differs from oxyhæmoglobin and its modifications in that its spectrum is not especially characterised by any band. This cyanogen compound gives to the blood a bright red colour. It is a very stable compound, allowing a current of air to be passed through it without the removal of the hydrocyanic acid from its union with the methæmoglobin; it also resists the reducing influence of the tissues, and may be recognised in the blood after death. According to Kobert, hydrocyanic acid renders the red blood corpuscles useless as oxygen carriers, and the bright red colour of the cyanomethæmoglobin accounts for the peculiar reddish tint of the post-mortem stains, and for the bright red colour of the mucous membrane of the stomach which is so frequently seen at the post-mortem examination in cases of hydrocyanic acid poisoning.

Treatment and antidotes.—The treatment of a case of hydrocyanic acid poisoning, if any signs of life exist, must be prompt and immediate. Cold water should be dashed on the face and back of the neck; smelling salts, or solution of ammonia, should be applied to the nostrils, and artificial respiration should be at once resorted to. If possible, vomiting should be induced by the administration of an emetic of mustard and water, or the stomach should be emptied by means of the stomach-pump; but generally it is impossible to empty the stomach by either of these means, on account of the firm clenching of the jaws. Brandy should be administered either by the mouth or rectum, ether injected subcutaneously, and friction applied to the extremities. If a battery be at hand, faradisation of the phrenics and diaphragm may be resorted to. An antidote consisting of sodium carbonate, ferrous sulphate, and ferric chloride, stirred together with water, has been suggested, with the object of converting the hydrocyanic acid in the stomach into prussian blue. This antidote is, however, useless, as there is no time for the preparation of so complex a mixture, considering the quick absorption of the poison, and the rapidity with which death occurs. The hypodermic injection of atropine has been suggested as an antidote, the suggestion being first

made on account of the well-known action of atropine as a powerful stimulant to the respiratory centre. A case has been recorded [1] in which a man took a considerable quantity of cyanide of potassium in solution (the actual amount is not mentioned) for the purpose of committing suicide, and, in order to be quite sure of the attainment of his object, he also swallowed a solution of atropine. Recovery took place, and on the following day he was quite well, although no other antidote had been given. From experiments made by Krohl [2] on dogs and cats, hydrogen peroxide appears to be an antidote to hydrocyanic acid, the apparent action being the oxidation, by the hydrogen peroxide, of the hydrocyanic acid to oxamide. These experiments show that hydrocyanic acid in larger quantities than the fatal dose, when administered to dogs and cats, had its effects stayed by means of hydrogen peroxide, and, moreover, the hydrocyanic acid could not be detected in the urine after the employment of hydrogen peroxide, whereas it is present in ordinary cases of hydrocyanic acid poisoning.

Post-mortem appearances.—The skin is either livid, or of a peculiar violet colour; the latter condition has been especially noticed in cases in which the quantity of the poison taken has been great, and it is more or less transient. The post-mortem stains are of a pinkish colour, due to the bright colour of the cyanogen compound with the blood. The nails are generally blue, the fingers clenched, the jaws firmly closed, and usually there is froth about the mouth. The eyes are wide open and glistening, and the pupils are dilated. Internally the odour of the hydrocyanic acid is usually marked on opening the stomach, thorax, and skull. The mucous membrane of the stomach is congested, and may be of a brilliant red colour. The blood is always fluid, and is frequently a bright arterial red, but it may be dark in colour.

Fatal dose.—The smallest fatal dose recorded [3] is half a drachm or thirty minims of the B. P. acid, equal to six-tenths of

[1] *Pharmaceutische Zeitung*, 1890.

[2] *Arb. Pharmakol. Inst. Dorpat*, 1892.

[3] *The Lancet*, 1888

a grain of the anhydrous acid. Recovery has taken place after taking half an ounce of the B. P. acid,[1] equal to 4·8 grains of the anhydrous acid; in this case the stomach-tube was quickly employed, along with other energetic treatment. In another case[2] two drachms, equal to about 2·5 grains of the anhydrous acid, were taken and recovery took place. This case is remarkable in that the person (a woman) who swallowed the acid was able to rush upstairs and tell her master (a medical man) what she had done, previous to falling down insensible; the treatment that was employed was the immediate use of the stomach-pump, the administration of apomorphine, and the employment of electricity and artificial respiration.

Fatal period.—The shortest time in which hydrocyanic acid has proved fatal is two minutes; the longest recorded fatal period is an hour and a half.

Analysis and tests.—I. The odour of the acid is marked and characteristic, although some people are unable to detect it.

II. *Prussian blue test.*—This consists in adding to hydrocyanic acid caustic potash or soda, solution of ferrous sulphate, solution of ferric chloride, and finally hydrochloric acid, when the dark blue colour of prussian blue is produced. If the solution of hydrocyanic acid be very dilute, the iron salts should be added in very small quantities, and the colour which is at first produced is generally more of a green than a blue. On allowing the liquid to stand some hours the prussian blue settles as a blue precipitate, or the liquid may be poured on to white filter-paper, when the blue deposit is left as a very evident film on the surface of the paper.

III. *Sulphur test.*—This consists in the conversion of the hydrocyanic acid into ferric sulphocyanide, which is of a deep blood-red colour. This conversion is effected by adding to the hydrocyanic acid solution of ammonia, and then a few drops of yellow ammonium sulphide, and evaporating the mixture carefully to dryness. The residue is dissolved in distilled water

[1] *Internat. Jour. Med. Sciences*, 1890.

[2] *Brit. Med. Jour.*, 1890.

acidified with hydrochloric acid, and ferric chloride added, when the deep blood-red colour of ferric sulphocyanide is produced. The colour is discharged by the addition of a solution of mercuric chloride.

IV. Solution of nitrate of silver gives with a solution of hydrocyanic acid a white precipitate, which, if allowed to settle and the liquid poured off, is dissolved by strong boiling nitric acid.

The above-mentioned tests are described as they should be employed to detect hydrocyanic acid in the liquid form; with regard to their relative delicacy, the iron and sulphur tests exceed the silver test when employed for the detection of liquid hydrocyanic acid; but if employed to detect hydrocyanic acid in the form of vapour, the silver test is the most delicate one. It is employed by placing a drop of a weak solution of nitrate of silver on the concave surface of a watch-glass, which is then inverted over a beaker or flask containing the liquid, or the contents of the stomach, or the viscus to be tested for free hydrocyanic acid; if necessary, the evolution of the vapour is facilitated by immersing the vessel containing the substances in warm water. The presence of hydrocyanic acid manifests itself as a white line round the margin of the nitrate of silver drop, which gradually extends as a film over the whole of the drop; if the deposit of silver cyanide has formed slowly, it will, when examined under the microscope, be found to consist of delicate prismatic crystals (fig. 15). The vapour test employed in this way is extremely delicate (fig. 16), but if the material examined be undergoing putrefaction, the test cannot be used,

FIG. 15.—CRYSTALS OF SILVER CYANIDE PRODUCED FROM VERY WEAK HYDROCYANIC ACID (Magnified 340 diameters)

as the sulphuretted hydrogen resulting from the decomposition would produce a film of black sulphide of silver, which would entirely obscure the white cyanide of silver. The prussian blue and sulphur tests may also be employed as vapour tests even when the substances tested are in a state of putrefaction. The prussian blue test is worked by placing a drop of solution of caustic potash on the concave surface of a watch-glass, and then inverting it for a few minutes over a vessel containing the substance evolving the vapour of hydrocyanic acid; the watch-glass is then removed, and the drop is stirred with two or three drops of a mixture of ferrous sulphate, ferric chloride and hydrochloric acid, when the prussian blue is developed; only mere traces of the iron salts should be employed in this reaction.

FIG. 16.—CRYSTALS OF SILVER CYANIDE PRODUCED FROM THE HYDROCYANIC ACID OF A SINGLE APPLE-PIP (Magnified 340 diameters)

The sulphur vapour test is worked by employing a drop of ammonium sulphide on a watch-glass, in place of the caustic potash; this, after a few minutes' exposure, is removed, and the drop evaporated to dryness either on a water-bath or cautiously over a spirit-lamp, and then touched with a glass rod that has been dipped into a weak solution of ferric chloride and dilute hydrochloric acid, when the blood-red colour of ferric sulphocyanide is developed. This colour is discharged by the addition of a drop or two of solution of mercuric chloride.

V. Another delicate test for hydrocyanic acid is the *nitroprussid test*, which consists in adding to a solution of hydrocyanic acid a few drops of potassium nitrite, two or three drops

of ferric chloride solution, and dilute sulphuric acid until a light yellow colour is obtained. The liquid is then heated to boiling, cooled, solution of ammonia added to precipitate excess of iron, filtered, and one or two drops of a very dilute solution of the colourless ammonium sulphide added. A very minute quantity of hydrocyanic acid gives a bluish-green colour. A larger quantity gives a violet-red colour, which changes to blue, green, and finally yellow.

VI. If a dilute solution of starch be tinged with iodine, the bluish-black colour is discharged on addition of a minute amount of hydrocyanic acid. This test is, however, only of value as a confirmatory test, since other reducing agents will also discharge the colour of iodide of starch.

From organic substances liquid hydrocyanic acid may be obtained by distillation with tartaric acid, and the above-mentioned tests can then be applied to the distillate. This process is, however, open to the objection that hydrocyanic acid might be derived from non-poisonous cyanides, or from a sulphocyanide, such as sulphocyanide of potassium, which, in small quantities, is contained in saliva; and, since the latter is swallowed, minute quantities of potassium sulphocyanide may be present in the contents of a stomach. To obviate this objection, the contents of a stomach may be distilled with a substance evolving carbon dioxide, which gas will liberate hydrocyanic acid if present in the free state, or if potassium cyanide be present, but will not decompose potassium sulphocyanide nor potassium ferrocyanide and the non-poisonous double cyanides. The following is the process :—

Jacquemen's process for detecting hydrocyanic acid or potassium cyanide in the presence of non-poisonous double cyanides.— The contents of the stomach are mixed with a concentrated solution of sodium bicarbonate and distilled. The distillate is then examined by any of the tests previously described.

Another process is the following :—

Barfoed's process for detecting hydrocyanic acid and poisonous cyanides in the presence of non-poisonous double cyanides.—The

contents of the stomach are acidulated with tartaric acid and shaken with an equal volume of ether. The ether is removed by decantation, and contains the free hydrocyanic acid. It is then shaken with a dilute solution of caustic soda, and the latter, after standing, is drawn off by means of the separating funnel, and tested by any of the previously mentioned tests.

The amount of hydrocyanic acid present in the contents of the stomach or viscera may be estimated by distillation with tartaric acid, and the subsequent precipitation of the hydrocyanic acid from the distillate as silver cyanide, which is then collected and weighed.

Detection of hydrocyanic acid in the blood.—Blood containing the cyanogen compound with methæmoglobin may be tested in the following manner, as described by Kobert[1]:—

One c.c. of the blood is diluted with ninety-nine c.c. of distilled water, and one per cent. of solution of potassium ferricyanide is added drop by drop, with continuous shaking. If cyanogen be absent, the colour of the solution passes from red to yellow, owing to the formation of methæmoglobin, and shows the spectrum of the latter. If cyanogen be present in the blood, the colour becomes brighter red, and shows no absorption band in the spectrum.

Another method, also given by Kobert, for distinguishing blood which contains the cyanogen compound from normal blood, is based on the fact that the self-reduction of the blood is arrested by the presence of the smallest quantity of hydrocyanic acid. If blood contains cyanogen, a one per cent. solution retains its colour for some time; whereas, a one per cent. solution of normal blood becomes darker at the end of a few hours or days, and, in place of the oxyhæmoglobin spectrum, shows the spectrum of reduced hæmoglobin.

Late detection of hydrocyanic acid.—If the viscera or blood containing hydrocyanic acid have undergone putrefaction, the acid may have been completely converted into ammonium sulphocyanide by the ammonia and sulphuretted hydrogen

[1] *Apoth. Zeit.*, 1891.

produced during putrefaction. This ammonium sulphocyanide may be dissolved out of the dried viscera or blood by alcohol, the alcoholic solution filtered and evaporated to dryness, when the residue will give the characteristic blood-red colour with ferric chloride. In this way Stevenson has detected the poison in blood kept for a year after death from hydrocyanic acid poisoning.

Potassium Cyanide

This substance is largely used in photography and in connection with electro-plating. As a poison it is similar in its action to hydrocyanic acid, and the symptoms produced by it are the same as those detailed in connection with hydrocyanic acid (see p. 221); but, since the commercial potassium cyanide frequently contains a large quantity of undecomposed potassium carbonate, it may, when taken, exert a corrosive action from the presence of that substance, and so may cause softening or erosion of the mucous membrane of the lips, mouth, and stomach, and lead to the production of blood-stained mucus on the surface of the stomach.

Post-mortem appearances.—The same as those given in connection with hydrocyanic acid (see p. 223), with the possible addition of the corrosive effects mentioned above.

Fatal dose.—Five grains of potassium cyanide have caused death in a quarter of an hour; but recovery has taken place after the taking of forty grains of it in a solid form.

Tests.—The same as those for the detection of hydrocyanic acid, and, in addition, the tests for potassium.

Preparations containing Hydrocyanic Acid

Essential oil of bitter almonds.—This volatile oil is obtained by distillation of bitter almond seeds with water. It is not present in the seeds, but is produced from a substance named amygdalin, which undergoes decomposition by means of a ferment named emulsin in the presence of water. Both

amygdalin and emulsin are present in bitter almond seeds, and, by their interaction in the presence of water, the essential oil of bitter almonds and hydrocyanic acid are produced.

The essential oil of bitter almonds contains hydrocyanic acid varying in amount from five to fifteen per cent. As a rule, it is now freed from hydrocyanic acid before being sold as a flavouring agent. If the hydrocyanic acid remain in it, it is necessarily a very poisonous substance. Thirty drops of the oil have proved fatal to an adult in somewhat under half an hour. Mistakes have occasionally occurred from confusing it with almond oil, or oil of sweet almonds, which is a fixed oil obtained by expression from sweet almonds. *Essence of almonds* consists of one part of the essential oil to seven parts of rectified spirit.

Cases of poisoning from eating bitter almond seeds are very rare. Maschka records the case of a woman, aged thirty-one years, who ate a number of the seeds, calculated to weigh 1,200 grains, and to be capable of yielding about 2·5 grains of pure hydrocyanic acid. In about a quarter of an hour she became insensible, and death occurred within an hour and a half from the first symptoms.

Cherry-laurel water, which is obtained by distilling cherry-laurel leaves with water, contains about 0·1 per cent. of hydrocyanic acid.

Bitter almond water, obtained by distilling bitter almond seeds with water, also contains a small quantity of hydrocyanic acid.

The kernels of the following fruits yield hydrocyanic acid in small quantities on treatment with water, viz. peach, cherry, apricot, plum, and apple. In the unbroken state, apple seeds yield no hydrocyanic acid, and, if swallowed, pass through the body unchanged. The seeds of four average-sized apples yield by distillation one-fortieth of a grain of anhydrous hydrocyanic acid, the same amount being obtainable from one bitter almond seed. In fig. 16 are represented some crystals of silver cyanide produced from the hydrocyanic acid of a single apple-pip.

Alcohol

In a work on medical jurisprudence it is only necessary to describe acute alcoholic poisoning. In cases where a medical man is called to see a patient in a condition of profound coma, and where no history as to the causation of the insensibility can be obtained, the correct diagnosis may be extremely difficult, and a careful methodical examination, as detailed below, should be made. Alcohol is eliminated by the kidneys and lungs, and during its elimination by the lungs in the form of vapour it communicates to the breath a characteristic odour.

Symptoms.—The symptoms of a poisonous dose of alcohol generally come on in a few minutes, and are : (i) Confusion of ideas. (ii) Tottering gait. (iii) Giddiness, followed by stupor and coma. (iv) Should recovery from this stage supervene, vomiting generally occurs ; if recovery from this stage do not take place, vomiting is usually absent. (v) The pupils are generally dilated and fixed, but they may be contracted almost to pin-points, and remain in that condition nearly up to the last, as in a case described by Berry.[1] If the pupils are dilated, but contract under the influence of light, the sign is to be regarded as a favourable one. (vi) The odour of alcohol in the breath. This, however, is practically of but little use, since on the discovery of a person in an insensible condition the first action of bystanders is, as a rule, to pour brandy or some other stimulant into the mouth.

The rapidity of the onset of alcoholic insensibility is very much influenced by the degree of concentration of the alcohol taken. If the alcohol be taken in a diluted form, a stage of excitement precedes the stage of stupor ; but if taken in a concentrated form, and in a very large dose, profound coma may ensue in a very few minutes.

It is important to remember that alcohol may cause death

[1] *The Lancet*, 1893.

in one of the three following ways: 1. If a large quantity of concentrated alcohol, such as a pint of whisky or brandy, be swallowed at once, death may result in a few minutes from shock. 2. If a large amount be taken in divided doses, the administration of which is spread over several hours, death may be due to syncope, preceded by prolonged coma. 3. The patient may pass through a comatose state, and make an apparent recovery, and then, on rare occasions, may suddenly have a relapse and die.

Comatose symptoms, due to opium and other narcotic poisons, to cerebral hæmorrhage, to concussion of the brain, to uræmic coma, to diabetic coma, to post-epileptic coma, and hysteroid stupor, have not unfrequently been mistaken for those of alcoholic poisoning. The following table gives the points to which attention should be particularly directed in making the differential diagnosis. Hysteroid stupor would be detected by taking into consideration the age and sex of the patient, and the absence of the symptoms of insensibility. Post-epileptic coma rather resembles deep sleep than true coma, and is chiefly met with in young persons. If the rare condition known as *status epilepticus* occur, in which a series of fits succeed one another, with intervals of coma, the tongue should always be examined to see if it has been bitten by the teeth, an accident which is a common occurrence among epileptics.

Treatment.—The stomach should be emptied by means of the stomach-pump or tube; if neither of these be procurable, an emetic of mustard and water should be given, or a hypodermic injection of apomorphine administered. Cold water should be dashed upon the face and head, and hot strong coffee should be given as a cerebral and cardiac stimulant. If a medical man be called to a person in a comatose state, and doubt exist in his mind, after the examination of the patient, as to the cause of the coma, it is advisable to treat the case as one of cerebral hæmorrhage.

Post-mortem appearances.—Rigor mortis is usually well

—	Onset of symptoms	Nature of breathing	Appearance of face	Condition of pupils	Temperature
Alcoholic coma	History of gradual onset, unless a very large dose of strong spirit has been taken	Slow and somewhat laboured (snoring); odour of alcohol in the breath, which, however, is of but little diagnostic use	May be either flushed or pale	Either dilated or contracted	Normal or Subnormal
Opium coma	Gradual	Laboured and stertorous; may be of Cheyne-Stokes' type when a fatal issue is impending; odour of breath	Pale	Contracted	Generally subnormal
Coma from cerebral hæmorrhage	Rapid	Stertorous	Paralysis of cheek and mouth on one side; possible conjugate deviation of eyes	Dilated, except in cases of hæmorrhage into pons, when the pupils are contracted	Not as a rule elevated except in cases of hæmorrhage into pons, when it is frequently 103° or more
Coma from concussion of the brain	Immediate	Nothing characteristic	Pale, as a rule	Usually contracted	Usually normal or subnormal
Uræmic coma	Gradual	Sometimes stertorous, or of a hissing nature; may be simply slowed	Pale or dusky	Contracted	Normal or subnormal
Diabetic coma	Gradual	Slow and sighing, or occasionally gasping in character; peculiar odour of the breath	Generally pale	Nothing characteristic	Normal or subnormal

marked and prolonged, and the onset of putrefaction is slow. On opening the cavities of the body the odour of alcohol may be detected in the stomach, liver, lungs, and brain, but the smell is not necessarily present in cases of alcohol poisoning which have terminated fatally. The mucous membrane of the stomach is sometimes found congested or inflamed, and frequently presents a deep cherry-red colour, which at first sight might suggest the action of a metallic irritant poison such as arsenic. The red colour is not necessarily the same all over the lining of the stomach; it may be bright red in places and pale in others. The lungs are generally found congested, and the right heart and veins are usually filled with dark fluid blood. The brain and its membranes are generally congested, the vessels being filled with dark fluid blood, and extravasations are sometimes found.

Fatal dose.—This is very variable, and depends very much upon the degree of concentration and the quantity of alcohol taken at one time. For children under twelve years of age, an amount of spirit containing one to two fluid ounces of absolute alcohol would probably prove fatal; for adults an amount of spirit containing three to five fluid ounces of absolute alcohol would probably prove fatal if taken within a short period of time. A child has died from drinking two ounces of gin, and an adult from drinking half a pint of gin.

Fatal period.—Very variable.

Analysis and tests.—Alcohol may be separated from organic admixture by distillation; if the organic substances are acid in reaction, they should be neutralised by the addition of sodium carbonate previous to distillation. To the distillate the following tests may be applied.

I. *Iodoform test.*—If to a solution of alcohol a few drops of aqueous solution of iodine in potassium iodide are added, and then sufficient caustic potash or caustic soda to decolourise the iodine, the liquid, on warming, will yield a crystalline deposit of iodoform, which may be recognised by its smell or by the appearance of hexagonal crystals under the microscope. Other

substances, however, such as aldehyde and acetone, also give the iodoform reaction.

II. *Bichromate test.*—If a solution of alcohol be heated with a few drops of solution of potassium bichromate and some strong sulphuric acid, the odours of, firstly, aldehyde, and afterwards acetic acid, are evolved, and the colour changes from yellow to green.

III. *Nitrous ether test.*—If to a solution of alcohol some copper turnings, a few drops of nitric acid, and some strong sulphuric acid are added, and the mixture warmed, the characteristic odour of sweet spirit of nitre is evolved.

IV. *Acetic ether test.*—If to a solution of alcohol a few crystals of sodium acetate and some strong sulphuric acid are added, and the mixture warmed, the odour of acetic ether is evolved.

Chloral Hydrate

This substance is now extensively employed as a hypnotic, and many cases of poisoning, principally accidental or suicidal, but occasionally homicidal, occur from its use. Chloral hydrate diminishes, and in poisonous doses annuls, the reflex irritability from the spinal cord, and in large doses produces profound coma.

Symptoms.—(i) In poisonous doses, after a short interval, drowsiness comes on, then passes into profound coma, which may terminate in death. (ii) The respiration is slow and laboured, but it may be quick and shallow. (iii) The heart is considerably depressed, and the pulse is small and feeble. (iv) The face is generally livid or pale, but occasionally flushed, as in a case seen by Welch.[1] The pupils are generally contracted and insensible to light, but occasionally they are slightly dilated. (v) The surface of the body is cold and clammy, and the reflexes are absent.

Plummer[2] records a case of a boy, aged sixteen years, who took probably over one ounce of chloral hydrate; three hours

[1] *The Lancet*, 1891. [2] *Ibid.*, 1894.

later he was quite unconscious, and could not be roused ; seven hours after taking the poison his temperature rose to 100° F. ; at a period of thirteen hours it was 103° ; at thirty-one hours it was 104·6°, and thirty-nine and a half hours after taking the poison he died without having recovered the slightest consciousness, although energetic treatment had been resorted to throughout.

In cases of fatty disease and valvular affections of the heart, comparatively small doses of chloral may cause sudden death by producing paralysis of the heart. Chloral hydrate is, to a slight extent, eliminated unchanged in the urine, but the greater part of it is decomposed in the system, one of the products of decomposition—urochloral acid—being found in the urine.

Treatment and antidotes.—The stomach should be emptied and washed out either by the stomach-pump or stomach-tube. If neither of these be procurable, an emetic of mustard and water, or a hypodermic injection of apomorphine, should be administered. The face and head should be flicked with a wet towel in the endeavour to rouse the patient. Warmth should be applied to the body by means of hot bottles and warm blankets, and hot strong coffee should be administered. The hypodermic injection of four minims of *liquor strychninæ* is useful. Smelling salts should be applied to the nostrils, and artificial respiration resorted to, if necessary. Inhalations of oxygen, administered at intervals, have been recommended, and appear to be beneficial.

Post-mortem appearances.—There are no characteristic appearances of chloral poisoning. The lungs and heart are generally found in the condition met with in cases of death from failure of respiration and circulation. Fluidity of the blood has sometimes been observed.

Fatal dose.—The smallest dose of chloral hydrate that has proved fatal was in the case of a child, one year old, whose death was caused by three grains. The smallest quantity that is recorded to have killed an adult is ten grains, which produced death in an old lady of seventy. In another case twenty grains

caused the death of an adult in half an hour, and in another case thirty grains proved fatal. Recovery has taken place, however, after taking very large doses of chloral. Welch [1] describes a case of recovery in a woman after taking 360 grains of chloral. Holburton [2] describes a case of recovery in a gentleman after having taken 240 grains. In another case recovery took place after 420 grains had been taken in one dose.[3]

Fatal period.—The shortest time within which death has occurred is fifteen minutes, and the longest period that has elapsed before the fatal event is thirty-nine and a half hours.

A case of homicidal poisoning occurred in Manchester in 1889 and was the subject of a trial, *Reg.* v. *Parton* (Manchester Ass., 1889). The prisoner was convicted of having caused the death of his victim, an elderly man, by the administration of hydrate of chloral in beer. Probably only a small quantity of hydrate of chloral was given, with the object of stupefying the victim with a view to robbery; but, owing to the fatty condition of the victim's heart, death resulted in all probability from paralysis of the heart. Traces of chloral hydrate were found in the contents of the stomach.

Analysis and tests.—To extract chloral hydrate from the contents of the stomach, they should be digested for twenty-four hours with three or four times their volume of absolute alcohol rendered acid by sulphuric acid; the alcoholic extract is separated by filtration and evaporated. The residue is repeatedly shaken with petroleum ether to remove fatty substances, and is finally shaken with ordinary ether to extract chloral hydrate; by the evaporation of this ethereal extract chloral hydrate is left. To detect chloral hydrate in the urine, it should be acidified with sulphuric acid, and then agitated, first with petroleum ether, and finally with ordinary ether to extract the chloral.

I. A solution of chloral hydrate, when agitated with solution of caustic potash, and, if necessary, gently warmed, evolves

[1] *The Lancet*, 1891. [2] *Brit. Med. Jour.*, 1892.
[3] Dixon Mann's *Forensic Med. and Toxicol.*

the odour of chloroform. If the solution be a strong one the chloroform is precipitated in the form of fine globules, and appears as a white precipitate.

II. If to a solution of chloral hydrate a few drops of ammonium sulphide be added, the mixture, either quickly or in a short time, according to the amount of chloral hydrate present, becomes opalescent, and finally acquires a yellowish or reddish-yellow appearance, an amorphous precipitate finally settling to the bottom of the tube ; at the same time a peculiar odour is developed. This test is an extremely delicate one.

III. If to a solution of chloral some alcoholic solution of caustic potash and a few drops of aniline be added, and the mixture well shaken and then warmed, the extremely disagreeable odour of phenyl isocyanide is developed. This test depends upon the conversion of the chloral by the caustic potash into chloroform. A similar reaction is given with chloroform, iodoform, bromoform, and trichloracetic acid.

IV. If to a solution of chloral hydrate an aqueous solution of caustic potash, in which a little β-naphthol has been dissolved, be added, and the solution gently warmed, a blue colour is produced. This test also depends upon the conversion of the chloral by the alkali into chloroform.

If chloral hydrate be present in the contents of the stomach in very small amounts, in order to avoid confusing it with chloroform, it is advisable to first distil the contents of the stomach with sufficient tartaric acid to render them acid, when if any chloroform distils over it must have existed as such in the contents of the stomach. If the fluid in the distilling flask be then rendered alkaline with caustic potash, and again distilled, any chloroform vapour which then comes over must be derived from the decomposition of chloral hydrate. For the delicate detection of chloroform vapour, see p. 243. In the urine both the chloral and the urochloral acid possess the property of reducing Fehling's solution, and hence may be mistaken for sugar in the urine.

Chloroform

As a liquid, chloroform is neither a very active nor commonly used poison. Poisoning by liquid chloroform is generally the result of accident, or of attempted suicide.

Symptoms.—The symptoms that follow the swallowing of chloroform in a liquid form are similar to those produced by its inhalation, with the addition of those due to the irritant effect of the liquid on the mucous membrane of the stomach and intestines. (i) Vomiting generally occurs. (ii) Unconsciousness soon supervenes, and the patient becomes comatose. (iii) The face is cyanosed, and the surface of the body clammy. The pupils are generally dilated, and do not react to light. The breathing is slow and stertorous, and the pulse feeble and small. (iv) If death occur, it is due to paralysis of the respiratory centres, and possibly to paralysis of the heart, as an additional, or, as some observers believe, as a primary cause. (v) If the patient recover consciousness, diarrhœa may occur. The liver is sometimes enlarged, and a certain amount of jaundice may be present.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or tube, or, if neither of these be procurable, a hypodermic injection of apomorphine should be administered; the face and head should be flicked with a wet towel, and three or four drops of nitrite of amyl held to the nostrils at frequent intervals. Artificial respiration should be employed, and warmth applied by means of hot-water bottles and warm blankets.

Post-mortem appearances.—There are no characteristic appearances. If much liquid chloroform has been swallowed, there may be signs of irritation of the mucous membrane of the stomach and intestines. The blood is frequently found fluid and dark.

Fatal dose.—The smallest quantity of chloroform in the liquid form that has killed is one fluid drachm; this occurred in the case of a boy four years of age. The smallest recorded

fatal dose in connection with an adult is about four fluid drachms (Falck). On the other hand, recovery has taken place after the swallowing of much larger doses. On one occasion a man swallowed four fluid ounces of chloroform; he then walked a considerable distance, became comatose, with dilated pupils and stertorous breathing, and finally recovered in five days.

A case of death from swallowing liquid chloroform was the subject of a sensational trial in London in 1886, *Reg.* v. *Adelaide Bartlett* (C.C.C., April 1886).

Fatal period.—Death has occurred in three hours after swallowing chloroform, but usually twelve hours or more elapse.

In cases in which anæsthesia is produced by means of chloroform vapour mixed with air, death occasionally occurs, and in such cases, the fatal termination is generally much more rapid than in cases of death from swallowing it in the liquid form. Death has occurred within two minutes from the commencement of inhalation of chloroform, and twenty minims inhaled have proved fatal. There are certain important medico-legal points that may arise in connection with the administration of chloroform as an anæsthetic with which the medical practitioner should be acquainted. One is, as to whether persons in a natural sleep can be put under the influence of chloroform without being awakened, and thus be either robbed or chloroformed to death. If the sleep be very deep, it has been found by experiment that this effect can be produced, but not so if the sleep be slight or partial. Another point is, that in some cases of alleged robbery and rape, it has been stated by the alleged injured person that sudden insensibility was produced either by the application of an open bottle of chloroform to the nostrils, or by applying a handkerchief saturated with chloroform to the face, or by waving such a handkerchief in front of the face. To anæsthetise an adult, not in a sleeping condition, against his or her will, is not possible without a prolonged struggle; the mere waving of a handkerchief before the face of such an individual would certainly not produce sudden

insensibility. Adults are not commonly rendered insensible with chloroform vapour until after the lapse of from five to eight minutes, and the quantity required to produce insensibility in an adult is usually between three and four fluid drachms given in one-drachm doses.

With regard to the mode of death in connection with poisoning by chloroform, the Report of the work of the Second Hyderabad Chloroform Commission is interesting. The results obtained by them confirmed the results obtained by the first Commission, and show that when animals are anæsthetised by chloroform, or by ether, respiration fails before the heart does. In the cases of death from chloroform administered to animals, they found that chloroform poisoning causes paralysis of the respiratory centre, and then gradual death, the heart ceasing its action later. The facts deduced from their experiments are briefly stated as follows:—(1) When chloroform freely diluted with air is continuously given, a gradual fall of the blood-pressure occurs, provided respiration is not impeded, and provided the animal breathes quietly without struggling or involuntarily holding the breath. As this fall in blood-pressure continues, the animal first becomes insensible, then the respiration gradually ceases, and lastly the heart stops beating. However concentrated the chloroform may be, it does not cause sudden death or stoppage of the heart. (2) Chloroform has no power of increasing the tendency to either shock or syncope during operations. (3) Struggling during chloroform inhalation, or anything interfering with the breathing, such as holding the breath, or asphyxia, produces irregularities in the circulation, and in the action of the heart. (4) The Commission found that, if natural and regular respiration were insured without struggling, holding the breath, or any interference with the breathing, it was possible to give chloroform to the production of full anæsthesia with a gradual fall of blood-pressure, and unaccompanied by any irregularity of the heart or circulation. (5) When death occurs from chloroform, it is invariably due to an overdose. (6) The important factor in the safe administration of

chloroform is that the breathing be natural, and that struggling and any other form of respiratory embarrassment be avoided, and that to effect this, the chloroform must be administered in an open cone or cap which is at first held far enough from the patient's face to avoid causing him to hold his breath or to struggle, and into which he should be directed at first to blow after each inspiration. The cone or cap is then gradually brought nearer to the face, and eventually quite close to it. Pallor and loss of pulse are signs of great danger, and signify that the patient has not been breathing properly, or that he has been asphyxiated, or that the respiratory centre has been paralysed. The fall of the blood-pressure ceases long before a dangerous point is reached, *if the inhalation be stopped, when the cornea becomes insensitive.* (7) In the administration of chloroform, the blood-pressure should fall regularly throughout the whole administration, and this regular fall of blood-pressure can only be insured by absolute regularity in the breathing; therefore, to keep the breathing regular, the main attention of the administrator is to be concentrated upon that point.

The experiments of McWilliams are opposed to those of the Commission, as he infers that chloroform has a direct influence on the heart, that death occurs from cardiac failure, and that the respiration may be continued even after the heart has stopped.

Treatment.—In cases of chloroform anæsthesia in which respiration ceases, or the pulse suddenly fails, artificial respiration should be immediately resorted to. The chin should first be drawn forcibly upwards and the tongue drawn forward. The face may be flicked with a wet towel, and nitrite of amyl applied to the nostrils. The hypodermic injection of four or five minims of liquor strychninæ is usually extremely beneficial.

Milne[1] makes the suggestion that, in cases in which there has been considerable loss of blood previous to the administration of chloroform, a hypodermic injection of strychnine before administering the anæsthetic, may be of use.

[1] *Brit. Mca. Jour.*, 1891.

Toxicity of chloroform.—By the repeated freezing of ordinary chloroform by means of intense cold, Pictet has prepared what he considers to be pure chloroform. The mother-liquor from which the chloroform has been frozen out resembles chloroform physically, but contains residues, which, according to Du Bois-Reymond, differ considerably physiologically, and have a deleterious effect upon the organism. As to what these impurities or residues consist of nothing is known, but it is possible that they are composed of bodies possessing a powerfully depressing action upon the heart. Chloroform may be derived from several sources, and many surgeons have maintained that bad results are apt to follow its use when it is obtained from certain makers. From experiments that have been made, it appears that these residues cause diminution of blood-pressure, and that respiration stops more quickly when chloroform containing them is used than when pure chloroform is employed. Du Bois-Reymond considers that pure chloroform will kill by failure of respiration, but that impure chloroform does so rather more rapidly. The amount of impurities, he admits, is very slight, but he believes that, although minute, they act strongly when dissolved in chloroform, and that their presence is enough to seriously affect the person or animal inhaling the drug.

Analysis and tests.—I. In cases of poisoning by chloroform, its odour may be detected in the stomach or lungs, but this method of detection is not to be considered a delicate one, nor is it likely to be of service if the viscera are in a state of putrefaction.

II. A very delicate method for the detection of chloroform is by the Ragsky process. If death has been caused by the inhalation of chloroform, the lungs should be selected for the operation of the tests. They should be finely minced, and mixed with a small quantity of water in a flask, the contents of which should be rendered slightly alkaline by means of sodium carbonate. The flask is provided with a double perforated cork (fig. 17); through one of the perforations a glass

tube open at both ends passes, so as to dip below the surface of the contents of the flask. Through the other perforation a piece of glass tubing bent at a right angle is passed. This is connected with a Bohemian glass tube which can be heated to bright redness over a broad bunsen-burner through a length of four to six inches, as shown in fig. 17. Four inches from the heated portion a strip of calico, one inch in width, is wound several times round the tube, and then allowed to dip into a

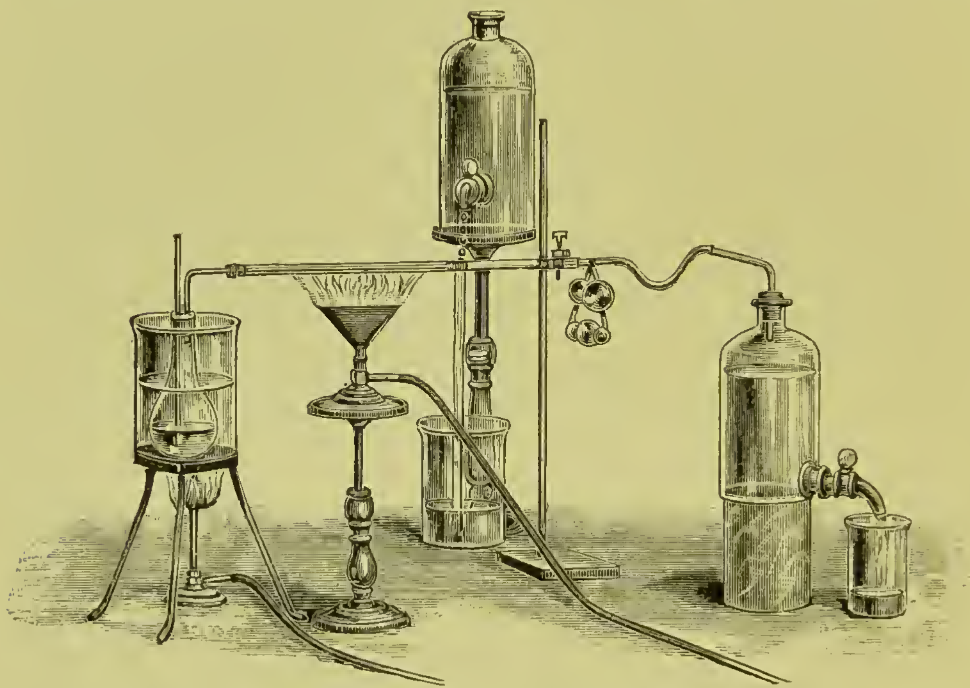


FIG. 17.—APPARATUS FOR THE DETECTION OF CHLOROFORM BY THE RAGSKY PROCESS

beaker; this is kept cool by water constantly dropping on it. In the part of the tube immediately in front of that around which the calico is wound, a piece of paper which has been dipped into a mixed solution of potassium iodide and starch is placed; in front of this the glass tube is connected with a set of Liebig's bulbs containing a solution of silver nitrate. These bulbs in their turn are connected with an aspirator. The flask is placed in a hot-water bath, and air is drawn through its contents,

and through the apparatus connected with it by means of the aspirator working at the extremity of the Liebig's bulbs; in this way any chloroform in the tissues contained in the flask is volatilised, and together with aqueous vapour passes through the red-hot portion of the tube, where it undergoes decomposition, with the production of chlorine and hydrochloric acid gases. The chlorine is detected by its turning the iodide of potassium and starch paper blue, and the hydrochloric acid gas is detected by its producing a white precipitate of silver chloride in the solution of silver nitrate contained in the bulbs. This method is an extremely delicate one for the detection of chloroform, and by this process Ludeking[1] has demonstrated that chloroform, when it has caused death by inhalation, can with certainty be detected in the body four weeks after death, and even when putrefaction has advanced to a very considerable extent. He also demonstrated that in cases of death not resulting from chloroform poisoning, the putrefied viscera gave no chloroform reaction by this method. The same process may also be applied to the detection of chloroform in the blood, and Stevenson has readily detected it in the blood of a person killed by inhalation of chloroform, when the body was examined twenty-four hours after death.

III. Chloroform may be separated by distillation from organic admixtures. A portion of the distillate may be tested by adding a few drops of aniline and some alcoholic solution of caustic potash; on gently warming the mixture for a short time, the very disagreeable odour of phenyl isocyanide is evolved if chloroform be present.

IV. Another test is to add to a portion of the distillate a solution of a small quantity of β-naphthol which has previously been dissolved in an aqueous solution of caustic potash; on gently warming the solution a blue colour is produced if chloroform be present.

The amount of chloroform present in any of the viscera may be determined by introducing a weighed quantity of the

[1] *St. Louis Academy of Science*, 1886.

minced substance into a flask fitted in the same way as in fig. 17, and passing the vapour of the chloroform through a combustion tube ten inches long, which is filled for about three-quarters of its length with pure lime and kept at a red heat; the chloroform vapour is decomposed, and the chlorine unites with the lime, forming calcium chloride. At the end of the experiment the tube is broken into an evaporating dish containing dilute nitric acid, which dissolves the lime and the calcium chloride; the chlorine can then be precipitated as silver chloride, which is collected, dried, and weighed.

The reason that chloroform remains so long in the lungs and viscera after death is, according to Dubois, due to the fact that the chloroform vapour penetrates into the interior of the tissues, and there becomes substituted for normal water, the protoplasm absorbing the vapour of the anæsthetic and expelling a certain quantity of water.

Bromoform

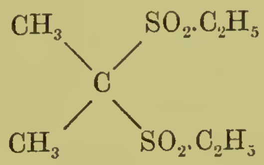
Dean [1] relates a case of poisoning which occurred in a child aged four years after taking between fifteen and twenty minims of bromoform. When seen half an hour later, the child was insensible, with pin-point pupils, livid, and breathing stertorously; the treatment consisted in emptying the stomach by means of the stomach-pump and emetics, and subsequent stimulation by means of external warmth, hot coffee and electricity. The child slowly recovered.

Sulphonal

This substance, which is now so extensively used as an hypnotic, is a derivative of methane (CH_4), from the molecule of which two atoms of hydrogen are displaced by two equivalents of methyl (CH_3), and the other two atoms by two equivalents

[1] *The Lancet*, 1893.

of the ethylsulphonic acid radical ($C_2H_5SO_2$). The constitution of sulphonal is, therefore, diethylsulphon-dimethyl methane; thus:—



Sulphonal

Sulphonal is produced by the oxidation of a mixture of ethyl-mercaptan and acetone. It occurs in white tabular crystals, tasteless and odourless, slightly soluble in water, but freely soluble in alcohol or ether. In large doses it is a poison.

Symptoms.—(i) Sleep, deepening into coma. (ii) Profuse perspiration. (iii) Slowing of respiration and circulation, and occasionally elevation of temperature, as occurred in a case described by Knaggs,[1] in which the temperature reached 103°. (iv) The urine is sometimes entirely suppressed, but more frequently is of a reddish-brown or reddish-black colour from the presence of hæmatoporphyrin. (v) Ataxic symptoms, muscular tremors, and vomiting have occasionally been observed prior to the advent of unconsciousness.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube, in the absence of which an emetic such as mustard and water, or a hypodermic injection of apomorphine, should be employed. Strong coffee may be administered as an antidote, or hypodermic injections of five minims of *liquor strychninæ* may be given at intervals.

Fatal dose.—Rather over one ounce of sulphonal has proved fatal to an adult.[2] Recovery has taken place after more than three ounces of sulphonal have been swallowed. If sulphonal be administered to persons suffering from considerable physical prostration, it may, even in medicinal doses, produce dangerous symptoms of increased prostration, and depressed action of the

[1] *Brit. Med. Jour.*, 1890. [2] *Ibid.*, 1890.

heart; this occurred in two cases described by Grant [1] in which single doses of twenty grains produced alarming symptoms of muscular weakness and prostration. Kober [2] describes a case of slow sulphonal poisoning in which sulphonal was given in doses ranging from seven to twenty-two grains (the latter dose only on a very few occasions) for a period of from four to five weeks. The urine gradually changed to a burgundy-red, and finally reddish-black colour. Retention of urine ultimately supervened and death occurred. Kober is of opinion that the sulphonal was stored away somewhere, possibly in the liver. Bresslauer of Vienna [3] has published a series of cases in which, out of twenty-seven insane patients who were treated with the drug for a considerable time, seven showed serious symptoms, and in five of these there was a fatal termination. The drug had been administered in good doses, and had been borne well until symptoms of disturbance set in; these were constipation, dark brown urine, feeble pulse, and great prostration. The cause of death was heart failure, with œdema of the lungs. Stern [4] found extensive necrosis of epithelium, and also minute hæmorrhages in the kidneys of a woman who died of coma following an attack of hæmato-porphyrinuria produced by taking sulphonal in daily doses varying from fifteen to thirty grains, and extending over a period of about three months, with, however, frequent intermissions.

Fatal period.—The shortest time in which sulphonal has proved fatal is three days.

Test.—If dry sulphonal be heated in a test-tube with reduced iron or charcoal, the odour of mercaptan is evolved.

Nitroglycerine

Nitroglycerine is a heavy oily liquid which produces by percussion a terrible explosion. When taken internally it exercises the general physiological action of a nitrite—viz.

[1] *Boston Med. and Surg. Jour.*, 1892. [2] *Centralbl. f. Klin. Med.*, 1892.
[3] *The Lancet*, 1891. [4] *Deut. Med. Woch.*, 1894.

relaxation of the arteries, accompanied by throbbing and sensation of fulness in the head, and frequently by violent pain in the head, lowered blood-tension, and accelerated action of the heart. In poisonous quantities it produces general paralysis, death being due to paralysis of respiration.

Symptoms.—After swallowing nitroglycerine the following symptoms may occur. (i) Burning sensation in the throat and œsophagus down to the stomach, followed by nausea and vomiting. (ii) Giddiness, flushing of the face, severe pain in the head, rapid action of the heart. (iii) Unconsciousness, stertorous breathing, cyanosis and general paralysis.

Fatal dose.—One ounce has caused death. Recovery has taken place after the swallowing of a tablespoonful of dynamite containing about two-thirds of its weight of nitroglycerine.

Fatal period.—After the swallowing of an ounce of nitroglycerine as referred to above, death occurred in four hours. After death from nitroglycerine the blood has been observed of a chocolate colour and containing methæmoglobin.

Carbon Bisulphide

Cases of acute poisoning from carbon bisulphide very rarely occur, but chronic poisoning from it is very common on account of its being largely used commercially in treating india-rubber, in extracting oils from seeds, and sometimes in the treatment of wool. In the cold process for vulcanising india-rubber, a mixture is used consisting of forty parts of bisulphide of carbon to from one to one and a half parts of chloride of sulphur. Cases due to acute poisoning by carbon bisulphide have been recorded in which amounts, varying from half an ounce to two ounces of the fluid, were swallowed.

Symptoms.—(i) Collapse, with coldness of the surface and cyanosis. (ii) Quick feeble pulse with laboured respiration. (iii) The peculiar odour of bisulphide of carbon may be detected in the breath, and also in the urine and fæces. (iv) If death occur, it is generally preceded by coma.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or tube. Stimulants and warmth should be applied to counteract the collapse. If the breathing be laboured, artificial respiration should be resorted to.

Post-mortem appearances.—Inflammation of the mucous membrane of the stomach has been found, together with the odour of the poison in the contents of the stomach. The veins have been found filled with black fluid blood; the action of the carbon bisulphide on the blood consists in the formation of methæmoglobin, with disintegration of the corpuscles.

Fatal dose.—About half an ounce has proved fatal; this case is recorded by Foreman.[1]

Fatal period.—Death has occurred in two hours and a quarter; this was the time noted in the case recorded by Foreman.

Chronic Poisoning by Carbon Bisulphide

Chronic poisoning or intoxication by bisulphide of carbon is not uncommon among workers in india-rubber factories, as the solution above referred to is used for the purpose of vulcanising. The first symptoms of bisulphide of carbon intoxication are great drowsiness and heaviness of the extremities, generally accompanied by intense headache in the forehead and temples, along with a marked decrease of the mental faculties. If the patient at this stage be sent into the fresh air he recovers very quickly. Later the appetite fails; there is headache, nausea, vomiting; the face becomes livid, and there is either mental excitement or depression. Sleeplessness is common, but the sufferer when asleep may have horrible dreams, and fancy himself surrounded by animals. While awake he may suffer from hallucinations resembling those of delirium tremens, and the intoxication from carbon bisulphide has not unfrequently been mistaken for that of alcohol. Later, symptoms of peripheral neuritis may supervene; these have been described by Ross,[2] and consist of tingling and numbness

[1] *The Lancet*, 1886. [2] *Med. Chron.*, 1887.

of the hands and feet, with progressive weakness and partial paralysis of the extensor muscles of the forearm and leg, producing respectively wrist-drop and ankle-drop. Petersen [1] describes three cases of acute mania from carbon bisulphide poisoning occurring in young men working in rubber factories. None of them presented any neuritis, and they all recovered. Rendu [2] reports a case of poisoning by carbon bisulphide occurring in a girl, aged fifteen, who was employed in vulcanising india-rubber. She had for some time suffered from frontal headache and heaviness of the head; the pains became more violent, and were accompanied by a painful contraction of the masseters, with stiffness of the neck and vertebral column; the legs and arms were subsequently attacked in a similar manner. Treatment with chloral, combined with removal from the source of poisoning, effected a cure in a few days.

Tests.—I. Carbon bisulphide can be detected after death in the blood, or during life in the saliva, by the phenyl-hydrazine reaction. Ten to fifteen c.c. of the blood are mixed with half their volume of water and distilled, and then treated in the same manner as described in connection with testing the saliva. Ten to fifteen c.c. of the saliva are shaken up with from twenty to twenty-five c.c. of benzene; this takes up the bisulphide of carbon, and on separating it and adding a few crystals of phenyl-hydrazine, a beautiful crop of crystals of a compound of the two substances deposits on standing.

II. Another delicate test is the triethylphosphine test. This consists in shaking the saliva, or the distillate from the blood, with an ethereal solution of triethylphosphine, when, if bisulphide of carbon be present, the liquid assumes a beautiful pink colour, due to the formation of a compound of the two substances.

[1] *Boston Med. and Surg. Jour.*, 1892.
[2] *Sem. Méd.*, 1891.

ALKALOIDS AND OTHER VEGETABLE POISONS

CHAPTER XV

General reactions of alkaloids—Extraction and estimation of alkaloids in viscera—Poisoning by opium—Preparations containing opium—Poisoning by morphine—Chronic poisoning by opium and morphine—Poisoning by codeine.

Vegetable alkaloids are organic bases resembling the alkalies in their properties of turning red litmus paper blue, and of uniting with acids to form salts; hence the derivation of the name 'alkaloid' from *alkali* and εἶδος, likeness. All alkaloids contain nitrogen, and are in fact derivatives of ammonia, the hydrogen of which has been partially or entirely displaced by various radicals. Chemically the vegetable alkaloids may be classified in three groups:—(1) those which are derivatives of pyridine, *e.g.* atropine, conine; (2) those which are derivatives of quinoline, *e.g.* narcotine, cinchonine; and (3) those which are substituted amines and amides. Most of the vegetable alkaloids belong to the first two groups. Alkaloids unite with acids to form salts. Most of the vegetable alkaloids are solid bodies; but a few, such as nicotine, conine and pilocarpine, are liquid. Animal alkaloids are basic substances obtained from animal tissues, and in some of their properties resemble the vegetable alkaloids; for the description of the animal alkaloids see pp. 397–404. Vegetable alkaloids possess several properties in common, among which are their capabilities of being precipitated from solutions by certain reagents which are called alkaloidal group reagents. Some of the alkaloids are precipitated by all of these reagents, others by only a few.

Alkaloidal group reagents—1. **Iodine dissolved in solution of potassium iodide. Wagner's reagent.**—This gives a reddish-brown or brown precipitate with most of the alkaloids, even when they are in very dilute solutions.

2. **Phosphomolybdic acid. Sonnenschein's reagent.**—This solution is prepared by dissolving phosphomolybdate of sodium in ten times its weight of water and adding one-tenth of its volume of strong nitric acid. This solution gives a yellow precipitate with nearly all the alkaloids. It, however, gives a precipitate with ammonium salts and ammonia derivatives, and also with salts of lead, silver, and mercury, unless excess of nitric acid be present.

3. **Potassio-mercuric iodide. Mayer's reagent.**—This solution is prepared by adding solution of potassium iodide to a solution of mercuric chloride until the red precipitate at first thrown down is redissolved in the excess of potassium iodide. Mayer's solution precipitates the majority of the alkaloids. The solution to be tested must not contain acetic acid. Other organic matters besides alkaloids are thrown down by Mayer's solution.

4. **Phosphotungstic acid. Scheibler's reagent.**—This reagent acts in a very similar manner to phosphomolybdic acid as a precipitant of alkaloids.

Tests for the individual alkaloids will be found in the sections devoted to them.

Extraction and Estimation of Alkaloids in Toxicological Enquiries

Extraction of alkaloids from the contents of the stomach or from the viscera can only be accomplished with great care and by a somewhat lengthy process. The points especially to be borne in mind in connection with their extraction and purification are, that they should not be brought in contact with strong mineral acids, nor subjected to a high temperature during the various steps of their extraction, since under either of these

conditions many of the alkaloids undergo decomposition. The best method for the extraction and estimation of alkaloids in toxicological enquiries is Stevenson's modification of the Otto-Stas method,[1] of which the following is an extract:—The viscus or organic material to be operated upon, if solid, is divided as minutely as possible, and is then digested with twice its weight of rectified spirit or rectified methylated spirit at a temperature of 35° C. If the organic material be liquid, it is treated with twice its volume of rectified spirit, and after twenty-four hours' digestion the liquid is poured off from the deposited solid, and the digestion repeated with a fresh quantity of spirit; this is again poured off and mixed with the first alcoholic infusion, the solid matter, if necessary, being squeezed in a piece of fine cambric. The solid undissolved matter is then again digested at 35° with rectified spirit faintly acidified with acetic acid; after twenty-four hours' digestion the liquid is strained off and the digestion repeated with unacidified spirit, the exhaustion with this spirit being continued so long as any colour is imparted to the spirit. Two or three digestions with unacidified alcohol generally suffice to accomplish this. The alcoholic liquids obtained before acidification are mixed together and rapidly raised to a temperature of 70°, at which they are only to be kept for a moment or two; they are then quickly cooled, filtered from matters that may have coagulated, the coagulated residue being washed with spirit. The liquids obtained with the acidified alcohol, and after the use of the acidified alcohol, are mixed together and similarly treated. These two alcoholic extracts are not mixed till a later stage is reached. They are separately evaporated at a temperature not exceeding 35° C. to the consistency of a syrup, the acidified extract being partly neutralised from time to time by the addition of a little caustic soda, so as to keep the liquid just perceptibly acid. The syrupy extracts so obtained are each well stirred in a small mortar with 30 c.c. of absolute alcohol, which is then poured off, and the pasty mass is again stirred up with successive quantities of

[1] Watt's *Dict. of Chem.*, vol. i., 1890.

15 c.c. of absolute alcohol as long as a colour is imparted to the alcohol. The alcoholic liquids are then mixed, filtered, the filter washed with alcohol, and the filtrate evaporated at a temperature not exceeding 35° C. The syrupy residues so obtained—viz. the one from the plain and the one from the acidified spirit—are diluted with a small quantity of water, filtered, the filters washed with water, and the filtrates mixed. These should together measure from 15 to 20 c.c.; this liquid will contain the whole of the alkaloids, and will be free from albumenoids, the latter having been coagulated during the temporary raising of the alcoholic liquids to 70°. This faintly acid aqueous liquid is transferred to a small separating funnel and twice its volume of washed ether added to it; the whole is then mixed by gentle agitation, care being taken not to emulsify the mixture by violent agitation. The ether is then allowed to separate, and the liquid below it is removed by means of the tap in the separating funnel (fig. 18) into another separating funnel, in which it is again agitated with a fresh quantity of ether. The liquid settling to the bottom is again withdrawn from the ether, and the operation of extraction with ether continued till a few drops of the ether on evaporation leave no residue; as a rule, four or five extractions suffice. This agitation of the faintly acid aqueous liquid with ether is to remove any fatty acids or oily matters that may be present, the alkaloidal salt or salts not being removed by agitation with ether. The ethereal solutions, before they are removed from the separating funnels, are suc-

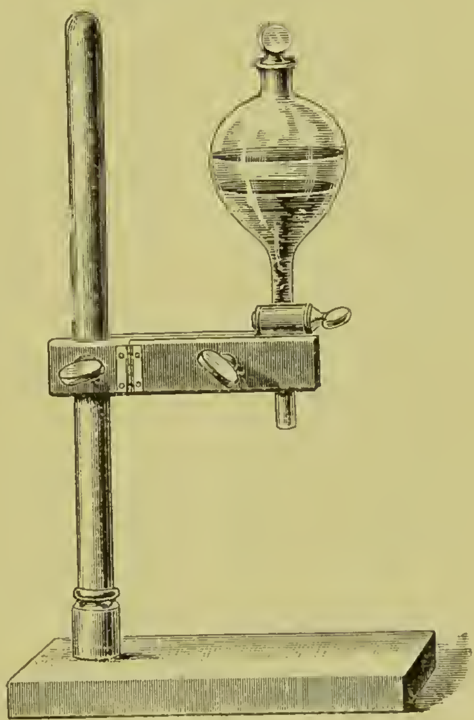


Fig. 18.—Separating Funnel

cessively washed by vigorous shaking with 5 c.c. of water, to which a few drops of dilute sulphuric acid have been added; this acidified water should be kept. This precaution is necessary, as some of the salts of the alkaloids are slightly soluble in ether, but by this expedient such traces are removed from the ethereal washings.

The acid aqueous solution and the water acidulated with sulphuric acid with which the ether has been washed are mixed, rendered alkaline with sodium carbonate, and then exhausted by agitation with, firstly, twice their volume of a mixture of one volume of chloroform and three volumes of ether, and subsequently with ether alone three or four times. These successive ethereal extracts are together put into a separating funnel, and are first washed by shaking with 5 c.c. of water, which on falling to the bottom is drawn off, and next shaken with 10 c.c. of water acidulated with sulphuric acid, the acid liquid being drawn off on separation, and lastly with 5 c.c. of water alone. By the alkalisation with sodium carbonate, the alkaloids are liberated from their salts, and then on agitation with the chloroform ether and ether they are dissolved by these solvents. The subsequent agitation with water acidulated with sulphuric acid converts them into sulphates, which, being insoluble in ether and chloroform, are removed in the acidified water, whilst at the same time impurities are left behind in the ether. The acid liquid and the final water-washing are then mixed, washed once or twice with a little ether, the ether removed, and the liquid realkalised with sodium carbonate, and well re-extracted with chloroform ether and ether. These ethereal solutions are removed by means of a separating funnel, and are washed with water rendered faintly alkaline with sodium carbonate. The ethereal solution is filtered through a dry filter, evaporated to dryness at a temperature a little below 35°C. in a glass basin, then dried for a few minutes at 100°C., and, after cooling over sulphuric acid, weighed; the weight will represent that of the alkaloids. Before evaporating the bulk of the solution, it is well to evaporate a few c.c. only,

and notice if an oily odorous residue be left, which would indicate the presence of a volatile alkaloid, such as nicotine or conine, in which case the evaporation must be modified by mixing the chloroform ether extract with sufficient ether, previously acidulated by agitation with strong hydrochloric acid, as is necessary to render it acid; the free alkaloids will thus be converted into their non-volatile hydrochlorides, which on evaporation of the chloroform ether are left and can then be weighed.

If a solid free alkaloid has been obtained, before applying the tests it should be converted into a hydrochloride by moistening it with a very slight excess of very dilute hydrochloric acid, and then evaporating to dryness in a vacuum over sulphuric acid. The dried residue may then be dissolved in water and the various tests for alkaloids applied to it. Of all the various alkaloids, morphine is the only one that would not be capable of extraction, except in very minute amounts, by this process. To obtain morphine, the first alkalised solution from which the other alkaloids have been removed must be re-extracted a few times with a washed mixture of equal volumes of acetic ether and ordinary ether, in which mixture morphine is soluble. The mixed acetic ether and ordinary ether extract is then washed with a little water, filtered through a dry filter, and evaporated in the manner previously described. The residue can then be examined for morphine. In place of mixed acetic ether and ether, hot amyl alcohol may be employed for the extraction of morphine, but amyl alcohol is an extremely unpleasant substance to work with, and the acetic ether and ether mixture is preferable.

Dragendorff has devised a method of separating mixed alkaloids in aqueous solution by using different solvents in sequence, one solvent taking up certain alkaloids, another solvent taking up others. Dragendorff's process consists in shaking the acid aqueous solution containing the mixed alkaloids successively with petroleum spirit, benzene, and chloroform, then alkalising it, and repeating the operation with the same solvents.

The substances of toxicological interest that may be so separated are the following:—From the acid solution *benzene* removes caffeine, colchicine, santonin, digitalin, and cantharidin. *Chloroform* removes papaverine, narceine, and picrotoxine. On rendering the solution alkaline with ammonia, *petroleum spirit* removes strychnine, brucine, veratrine, aconitine, emetine conine, nicotine, lobeline, and aniline. *Benzene* further removes atropine, hyoscyamine, physostigmine, codeine, narcotine, and additional quantities of strychnine, brucine, aconitine, veratrine, and emetine. *Chloroform* removes from alkaline solution some morphine, and additional quantities of narceine and papaverine. Amyl alcohol finally removes from alkaline solution some morphine, and additional quantities of narceine.

Opium and Morphine

Opium is the inspissated or naturally dried juice obtained by making incisions into the unripe capsules of the *Papaver somniferum* (Nat. Ord. Papaveraceæ), the *opium poppy* (Plate I.). A spiral incision is generally made into the unripe poppy capsule while it is on the stalk, the incision not being allowed to penetrate into the interior, as, in such a case, the juice would run inside and be lost. The juice, after it has exuded and partially dried, is scraped off, and is then allowed to further dry by exposure to the sun; the partially dried portions are mixed together into lumps which constitute the opium of commerce. Several alkaloids are present in opium, of which the most important are morphine, codeine, narcotine, narceine, papaverine, and thebaine. Of these, morphine is by far the most important alkaloid, and the one to which opium mainly owes its toxic properties. Morphine exists in opium partly as a sulphate, and partly as a meconate. From the medico-legal point of view, morphine and meconic acid are the two important constituents of opium, since, in the analytical detection of opium in cases of suspected poisoning, the analysis is narrowed down to the discovery and identification of these two bodies. As meconic acid is not met with in

Plate I.

Poppy (Papaver Somniferum)

any other substance but opium, the detection of it is generally considered to furnish sufficient indication of the presence of opium. Good opium should contain not under ten per cent. of morphine.

Acute Poisoning by Opium or Morphine

Symptoms.—These usually commence from half an hour to an hour after taking the poison, if taken by the mouth, but may come on much sooner if the morphine be injected hypodermically. The symptoms generally are—(i) A short period of pleasurable mental excitement, accompanied by a flushed face and increased brilliancy of the eyes. This stage occurs from a few minutes to half an hour after taking the poison, and is due to excitation of the cortex of the brain. (ii) Symptoms of cerebral depression next supervene; these are depression and lassitude, headache, or a feeling of oppression in the head, drowsiness, stupor, and complete insensibility, which may pass on to coma, accompanied by stertorous breathing. Previous to the advent of complete insensibility, the patient can be roused for a short time by a loud noise, but speedily relapses into a state of stupor; when, however, the comatose stage is reached, the patient is incapable of being roused by any noise. (iii) The respiration is at first hurried, afterwards it becomes laboured and irregular, and in the comatose stage it is stertorous. If a fatal termination be approaching, the breathing may become more embarrassed, and may assume the Cheyne-Stokes type. (iv) The skin is cold and moist, as opium and morphine arrest all secretions except that of the skin, the latter being generally excessive. There is a general relaxation of the muscular system. (v) The pupils are in the early stages contracted, and are insensible to light. In the last stage of opium or morphine poisoning they may be dilated, and also insensible to light; this dilatation of the pupils is generally a fatal sign, being indicative of a widespread muscular paralysis. (vi) Vomiting, and even purging, may very occasionally occur, but as a rule constipation results from opium poisoning. It is a favourable sign if

vomiting take place before the occurrence of stupor. (vii) The odour of opium may be noticeable in the breath, if opium or one of its preparations, such as the tincture, liquid extract, &c., has been taken. In cases of poisoning by morphine and its salts, no such odour would be present.

Very occasionally symptoms of an exceptional character occur. These are a quickened pulse, spasms or convulsions of a tetanic character, and dilatation of the pupils in the earlier stage; or very rarely hyperpyrexia is produced, as occurred in a case described by McNicol and Angus,[1] in which a man, aged twenty, took ten drachms of tincture of opium containing not less than forty grains of opium. Fourteen hours later his temperature rose to 109°, and he died of cardiac failure brought on by the hyperpyrexia.

The signs that are indicative of an approaching fatal termination in cases of poisoning by opium or morphine are an extremely feeble pulse, dilated pupils unaffected by light, general muscular relaxation, as indicated by dropping of the limbs, wrist-drop, falling of the lower jaw, and relaxation of the sphincters (including the sphincter iridis). Frequently persons have recovered from the severe symptoms of opium or morphine poisoning, and then after several hours have had relapses, with coma and fatal terminations. The same has very occasionally happened even after an interval of days.

Hæmorrhage into the pons varolii has been mistaken for opium poisoning, on account of the conjunction of insensibility with contracted pupils. Carbolic acid poisoning has occasionally been mistaken for opium poisoning, as the pupils are contracted, and there is generally coma and stertorous breathing. In most cases, however, the odour of carbolic acid in the breath would prevent such a mistake in diagnosis.

Treatment and antidotes.—If the poison has been swallowed, the stomach-pump or stomach-tube should be employed, and the stomach well washed out. If neither of these be procurable, vomiting should be promoted either by giving an emetic of

[1] *Brit. Med. Jour.*, 1893.

mustard and water, or by hypodermic injection of one-tenth of a grain of apomorphine, or by tickling the fauces. Strong hot coffee should be freely administered, and the faradic current may be usefully employed, by means of the wire brush if possible. The squirting of small quantities of cold water at intervals on to the lips and nostrils by means of a Higginson's or other syringe is a useful method of rousing the patient, but copious cold water douches are better avoided, as tending to render the surface too cold, and so adding to the collapse. The walking about of the patient, while from time to time he is shouted at and shaken, is not to be recommended, although it is advised in many text-books; such treatment has a great tendency to exhaust the vital powers. The dragging about of a patient in a comatose condition is worse than useless. If deeply comatose, artificial respiration should be resorted to, and faradisation of the phrenics may also be employed. Smelling salts should be held to the nostrils from time to time. Merry[1] recommends the inhalation of oxygen in opium poisoning when accompanied by intense cyanosis, dyspnœa, and a flagging pulse; the inhalation should be continued for twenty minutes, and repeated, if necessary, at intervals of half an hour. As an antidote four minims (one twenty-fifth of a grain) of solution of atrophine sulphate should be hypodermically injected, and the dose repeated until half a grain has been given.

For many years there has been much speculation as to whether atropine is a physiological antidote to morphine. Certainly their physiological actions are in nearly all cases opposite, for morphine contracts the pupil of the eye, atropine dilates it; morphine produces diaphoresis, atropine arrests the action of the sweat glands; morphine mostly constipates, atropine has the opposite effect; morphine may produce retention of urine, atropine the opposite effect; irritation of the skin, which is sometimes produced by morphine, is prevented by atropine. In cases of heart disease with engorged pulmonary circulation morphine is badly borne, whereas the

[1] *The Lancet*, 1894

addition of a small quantity of atropine does away with any disadvantages. But whether atropine is a physiological antidote to morphine or not, it is certainly a valuable and powerful stimulant to the respiratory centre, and undoubtedly it has proved of great value in many cases of poisoning by opium or morphine. It should be given in repeated doses, as mentioned above, until half a grain has been administered. Cruse[1] describes the case of an infant a week old who was accidentally poisoned by a grain of morphine. The comatose condition that resulted remained unaffected by various treatments for several hours until a quarter of a grain of atropine was administered and the dose repeated at an interval of half an hour. Recovery was complete in thirty-six hours, and no unpleasant symptoms attended the administration of such large doses of atropine. Atropine, however, has a tendency to be dangerous at one stage of opium poisoning, and perhaps strychnine is a safer antidote to employ, as it tends to avert the paralysing action of narcotic poisons generally on the respiratory centre. It is especially indicated where irregularity or interruption of breathing appears to threaten failure of the respiratory centre. Strychnine should be administered subcutaneously in doses of $\frac{1}{100}$ to $\frac{1}{50}$ of a grain (one to two minims of *liquor strychninæ*) according to the age of the patient, the dose being repeated at intervals of an hour two or three times. If respiration fail, artificial respiration must be resorted to, and if the circulation threaten to fail, inhalations of nitrite of amyl must be given. Picrotoxine has been recommended by Bokai[2] as an antidote for morphine, on the grounds that it exerts a powerful stimulating effect on the respiratory centre, and also that it is a powerful stimulant to the vaso-motor centre, whereas morphine may produce such rapid reduction in blood-pressure as to endanger life.

Post-mortem appearances.—With the exception of the smell of opium, which may be detected (although it is not necessarily present in cases of opium poisoning) in the contents of the stomach or intestines, there are no characteristic post-mortem

[1] *Archiv. für Kinderheilk*, 1893. [2] *Internat. Klin. Rundschau*, 1889.

signs of opium or morphine poisoning. Congestion of the vessels of the brain and its membranes is frequently seen, and there may be fluid in the ventricles of the brain. Hyperæmia of the lungs, such as would be produced by death from asphyxia, may or may not be present.

Fatal dose of opium.—The smallest dose that has proved fatal in the case of an adult was a quantity of extract of opium, equivalent to four grains of opium. Two fluid drachms of the tincture have also proved fatal—a quantity which would contain about eight grains of opium. Disease of the kidneys predisposes to the fatal effect of opium, so that in the case of a person suffering from kidney disease a much smaller dose than that mentioned above might prove fatal. The author has seen a case in which fifteen minims of the tincture of opium, containing one grain of opium, proved fatal to an adult suffering from Bright's disease. On the other hand, recovery has taken place after three ounces of the tincture, containing ninety-nine grains of opium, had been taken [1]; and, in a case recorded by Boustead,[2] recovery took place after eight ounces of laudanum had been swallowed by a woman. It is important, however, to bear in mind that infants and children are peculiarly susceptible to the effects of opium, and very small quantities may produce in them fatal results. Taylor and Stevenson record the following cases:—

1. A child, aged nine months, was killed by four minims of laudanum (equal to a quarter of a grain of opium).
2. An infant, five days old, was killed by two minims of laudanum (equal to one-eighth of a grain of opium).
3. An infant, seven days old, was killed by one minim of laudanum (equal to one-sixteenth of a grain of opium).
4. An infant, four weeks old, was killed by a dose of paregoric containing one-ninetieth of a grain of opium.

The latter is the smallest dose of opium on record that has proved fatal, but there is some ground for doubt as to whether so extremely minute a dose could produce a lethal result. On

[1] *Dublin Jour. of Med. Science*, 1892. [2] *The Lancet*, 1873.

the other hand, recoveries have taken place after infants and children have swallowed much larger doses of opium, especially if vomiting has occurred, or has been induced soon after the taking of the poison, and if artificial respiration has been resorted to, and, if necessary, kept up for some time. For instance, an infant, six days old, after swallowing a powder containing a grain and a half of opium, recovered after artificial respiration had been kept up for three hours. In another case an infant, three months old, recovered after swallowing a teaspoonful of laudanum, vomiting occurring shortly after it was swallowed. Morgan[1] treated an infant, aged one month, to whom three drops of laudanum had been administered; artificial respiration was kept up almost constantly for three hours, and recovery finally took place after forty-five hours. Among adults, recovery has constantly taken place after very large doses, such as several ounces of the tincture of opium or laudanum, have been taken.

The external application of opium, especially to an open sore or abraded surface, has caused death, the occurrence being more common in infants. Christison mentions a case in which a laudanum poultice applied to the abdomen of an infant produced fatal narcotism after some hours, and at the post-mortem examination a strong smell of opium was noticed within the body.

Opium habit.—As is well known, the human system can rapidly acquire a remarkable tolerance for large doses of opium; this was shown in the case of De Quincey, who brought himself to the daily use of nine ounces of laudanum, which is equivalent to about 360 grains of opium.

Fatal period.—The shortest time in which opium has produced a fatal result is three-quarters of an hour. The usual period is from six to twelve hours; the longest fatal period that has been recorded is twenty-four hours.

Preparations containing opium.—*Laudanum*, or tincture of opium, contains one grain of opium in fifteen minims, or nearly thirty-three grains of opium in one fluid ounce, which is equi-

[1] *Brit. Med. Jour.*, 1888.

valent to about 3·3 grains of morphine in the fluid ounce. *Paregoric*, or compound tincture of camphor, contains one grain of opium in half a fluid ounce. *Dover's powder*, or the compound powder of ipecacuanha, contains one grain of opium in ten of the powder. *Chlorodyne* contains about four grains of morphine in one fluid ounce, and, in addition, chloroform and hydrocyanic acid are present. *Syrup of poppies* is, if properly prepared, a decoction of the poppy capsules sweetened with sugar; as sold, it frequently consists of simple syrup, to which a variable amount of laudanum has been added. Taylor and Stevenson mention two fatal cases from the use of syrup of poppies, one in which an infant, five weeks old, was killed by three-quarters of a teaspoonful of the syrup, and another in which a child, eighteen weeks old, died from the effects of a teaspoonful; it is always a most uncertain preparation as regards its strength. *Battley's solution*, or sedative solution of opium, is an aqueous solution of opium, to which a little spirit has been added. It is generally considered to be stronger than the tincture of opium; twenty drops of it have proved fatal to an old woman, and a drachm and a half of it proved fatal on one occasion to a lunatic (age not mentioned). *Godfrey's cordial* is a mixture of tincture of opium, infusion of sassafras, and treacle; half a teaspoonful of it is said to have caused the death of an infant. *Nepenthe* is an alcoholic solution of meconate of morphine, and is of about the same strength as laudanum. *Black drop* is essentially an acetic acid solution of the constituents of opium, and is usually considered to be about four times as strong as laudanum. The original process of making it consisted in extracting opium with verjuice—the juice of the wild crab. *Winslow's soothing syrup* is said to contain nearly one grain of morphine, with other opium alkaloids, in an ounce ('Pharm. Jour.' June 1872, p. 975).

Morphine and its Salts

The symptoms of morphine poisoning are the same as those of opium, but convulsions are more common than in connection

with opium poisoning, occasionally these convulsions are of a tetanic character, and might at first suggest the presence of strychnine. Death from the hypodermic injection of morphine is now of very common occurrence, and, when so administered, it is more speedy in its action than when the drug is taken internally. Morphine has proved fatal after being sprinkled on an open sore.

Elimination of morphine.—Morphine is eliminated to a great extent by the bowels, to a lesser extent by the kidneys, and to a still slighter extent by the saliva. Tauber [1] gave a dog frequent hypodermic injections of morphine in small doses, and from the animal's fæces he recovered 41·3 per cent. of the injected morphine. Alt [2] made a series of experiments as to the excretion by the stomach of morphine injected subcutaneously. His experiments showed that after hypodermic injections of morphine the drug is largely excreted by the stomach, that this excretion begins perceptibly two minutes and a quarter after injection, persists distinctly for half an hour, and gradually ceases within an hour. After hypodermic injection of morphine, vomiting may begin when the morphine is being excreted by the stomach, and it is avoided by washing out the stomach. The quantity of morphine thus excreted by the stomach after subcutaneous injection may be estimated at half the amount injected. Symptoms of poisoning after subcutaneous injection of morphine are decidedly ameliorated by continued washing out of the stomach, by which means doses which would otherwise be fatal can be tolerated without injury. Dixon Mann examined the excretions of patients after taking large medicinal doses of morphine, and found that the alkaloid could always be detected in the fæces, and occasionally in the urine. Rosenthal [3] detected morphine in the saliva of patients after hypodermic injections; the reactions could not always be obtained immediately, sometimes not until the third or fourth day after injection; the

[1] *Archiv. für expt. Pathologie*, 1890.
[2] *Berliner Klin. Wochenschr.*, 1889.
[3] *L'Union Médicale*, 1893.

reaction sometimes persisted for several days, from which he inferred that the morphine probably, to some extent, accumulated in the system. This elimination of the morphine by the saliva has no connection with the similar function exercised by the stomach; Rosenthal found by experimenting on individuals who were instructed not to swallow their saliva, that after a short period morphine could be detected in the gastric juices of such individuals.

Decomposition of morphine in the system.—The question as to whether morphine undergoes decomposition in the organism is undecided. Lamal[1] considers that morphine is sometimes entirely, sometimes partially, converted into oxydimorphine, which may be eliminated in the urine. Tauber,[2] on the other hand, after adding a solution of morphine to blood, and circulating the mixture through the livers and kidneys of recently-slaughtered pigs, found that the morphine could be recovered entirely unaltered, and hence he concluded that it suffers no change in the body, and is therefore excreted unchanged. The objection to these experiments is that the circulation of the morphine in the blood through the livers and kidneys of dead animals is not comparable with its circulation through the viscera of living animals.

Fatal dose.—The smallest dose of a morphine salt that has proved fatal to an adult[3] is half a grain of the acetate of morphine, which proved fatal in from six to seven hours after it had been taken by the mouth. Reese mentions a case where three-quarters of a grain, administered hypodermically, proved fatal to a gentleman within twenty-four hours. One grain of morphine or of its salts has caused death on several occasions. The smallest dose of morphine that has killed an infant is one-twelfth of a grain of the hydrochloride of morphine.[4] On the other hand, recovery has taken place after very large doses of morphine, where effective treatment has been employed; thus

[1] *Jour. Pharm. Chem.*, 1889. [2] *Archiv. für expt. Pathologie*, 1890.
[3] *The Lancet*, 1838. [4] Taylor and Stevenson's *Med. Jurispr.*, vol. i.

recovery has taken place after thirty, thirty-six, fifty-one, and seventy-five[1] grains respectively of a morphine salt have been swallowed. Pope[2] records a case of recovery after the hypodermic injection of twelve grains of morphine; the patient was a girl, aged nineteen, and the dangerous symptoms lasted for thirty-six hours; the treatment consisted in the administration of four grains of atropine in $\frac{1}{20}$-grain doses at short intervals during twelve hours, artificial respiration, coffee, and the employment of various methods to rouse the patient.

Disease of the kidneys, especially the contracted granular form, predisposes to the fatal effect of morphine, and small doses which might be borne with impunity by a healthy person might in the case of an individual suffering from cirrhotic kidney prove fatal.

Chronic Poisoning by Opium and Morphine

The opium or morphine habit is one which, unfortunately, has become of late years very common. In many cases the habit has been acquired from the original use of opium or morphine for the relief of pain, and especially the employment of morphine by hypodermic injection has been acquired as a habit on account of the ease and rapidity with which the system is brought under the influence of the drug. The use of the drug produces an agreeable sensation at first, which, however, is followed by depression, to counteract which another, and generally an increased, dose is taken, and so the habit becomes acquired. Not only does the opium or morphine habit produce grave physical ill-effects as shown by dyspepsia, emaciation, intestinal troubles, and nervous symptoms which may resemble those of peripheral neuritis, but in addition moral degradation results, and the opium-eater or morphio-maniac will resort to all kinds of deception and lying in order to obtain the material to satisfy his desires. Enormous doses of opium

[1] *Am. Jour. Med. Sci.*, 1862. [2] *The Lancet*, 1894.

or morphine are consumed by those addicted to the habit. As previously mentioned, De Quincey had accustomed himself to take daily nine ounces of laudanum, equivalent to about 360 grains of opium, and forty [1] grains of morphine acetate have been injected daily for months. The only effective treatment is to stop the use of the drug at once, and for this purpose restraint of the individual may occasionally be necessary. If the sudden removal of the drug cause dangerous collapse, the rapid withdrawal of it in from six to twelve days may be employed.

Analysis and tests.—For the method of extracting morphine from organic mixtures see p. 257. The best solvent for the extraction of morphine is a mixture of equal parts of acetic ether and ether. As crystalline morphine is much less soluble in this solvent than freshly precipitated amorphous morphine, the mixture of acetic ether and ether should be poured upon the acid solution containing the morphine, and bicarbonate of soda solution added so as to produce alkalinity, after which the mixture is immediately well shaken. In this way the morphine, set free by the sodium bicarbonate, has no time to assume a crystalline condition before it is dissolved by the acetic ether and ether mixture.

I. If a drop or two of strong nitric acid be added to morphine in the solid state, an orange-red colour is produced. This colour remains unchanged on addition of stannous chloride, thus distinguishing it from the blood-red colour produced by the action of nitric acid on brucine (see p. 288).

II. Sulphomolybdic acid (Frohde's reagent), when rubbed with a small quantity of dry morphine, produces a violet or purple colour, changing to green and finally to sapphire-blue; this test is an extremely delicate one. It is the initial colour change—viz. the violet or purple colour—that is the characteristic one, the final change to blue being produced by several other bodies when brought in contact with sulphomolybdic acid.

[1] *Brit. Med. Jour.*, 1889.

The sulphomolybdic acid reagent is prepared by dissolving, with heat, six grains of ammonium molybdate or molybdic oxide in two fluid drachms of strong sulphuric acid; the reagent should be prepared fresh, as it deteriorates by keeping.

III. If ferric chloride solution be added to a neutral solution of a morphine salt, a bluish-green colouration is produced. This colour is due to the reduction of some of the ferric chloride to the ferrous state, and therefore on the subsequent addition of potassium ferricyanide a deep blue colouration is produced. This is a delicate test for morphine, in the absence of other substances which might reduce the ferric chloride. According to Armitage, one part of the morphine salt in 100,000 will give this reaction after standing a few minutes. It should be borne in mind that ferric chloride added to morphine meconate produces a deep red colouration, the blood-red colour of the ferric meconate completely masking the bluish colour produced by the action of the ferric salt on the morphine.

IV. If to morphine some strong sulphuric acid be added, and then a small crystal of potassium bichromate stirred in, a green colouration is produced.

V. Morphine liberates the iodine from iodic acid, but since albuminoid and other organic bodies also do the same, the reaction is not an absolute proof of the presence of morphine, but may be used as a confirmatory test. If to the solution of morphine, solution of iodic acid be added, a yellow or brown colour is produced; this brown colour is increased by adding ammonia. A delicate method of employing the iodic acid test is to convert morphine into a neutral salt with hydrochloric acid, and with a little of the solution mix a minute quantity of starch paste, and then evaporate to dryness at a gentle heat in a small porcelain dish; when the residue has cooled, a drop of a solution of one part of iodic acid in fifteen of water is added, when a blue colour will be developed if only the $\frac{1}{20000}$ of a grain of morphine be present.

VI. Iodised hydriodic acid gives with very dilute solutions of morphine salts a dark red precipitate. A somewhat similar

precipitate is given by many other alkaloids, so that this reaction is only of use as a negative test. If a neutral solution yield no precipitate with this reagent, morphine is not present.

Detection of Opium

The search for opium in organic mixtures is confined to the detection of morphine and meconic acid. The organic contents of the stomach, or any of the viscera, should be carefully smelt in order to ascertain if the odour of opium be perceptible. The solid contents of the stomach, or any viscus, should be cut into small pieces and reduced to a pulp by pounding in a mortar, or, if the matter be liquid, it should be evaporated to an extract. To the semi-solid matter rectified spirit acidulated with acetic acid is added, and the mixture digested for twenty-four hours at a temperature not exceeding 35°C. When cold, the mixture should be filtered through fine cambric, and the solid residue well washed with strong alcohol. The alcoholic liquid should be evaporated at a temperature not exceeding 35° C. to dryness, and the residue digested in absolute alcohol, which is then filtered and the filtrate evaporated to dryness. The dry residue is shaken up with warm water, filtered, and lead acetate added until a precipitate ceases to fall; the precipitate, which contains meconate of lead, is collected on a filter, whilst the filtrate will contain the morphine as morphine acetate. The precipitate of meconate of lead, after being washed, should be suspended in a small quantity of water, and decomposed by passing sulphuretted hydrogen through the mixture; this precipitates the lead as lead sulphide, and meconic acid is left in solution. The blackened mixture is then warmed to expel excess of sulphuretted hydrogen, filtered, the filtrate evaporated to dryness, and the residue tested for meconic acid. The test for meconic acid is that ferric chloride gives a deep blood-red colour of ferric meconate, which colour is not discharged on the addition of solution of mercuric chloride; whereas the corresponding blood-red colour of ferric sulphocyanide is

discharged by that reagent. The blood-red colour of ferric meconate is not discharged by the addition of dilute hydrochloric acid, whereas the red colour of ferric acetate is. The distinction of ferric meconate from ferric sulphocyanide is important, since small quantities of sulphocyanides are generally present in the contents of the stomach, partly due to swallowed saliva, which contains a minute quantity of potassium sulphocyanide, and partly due to the consumption of mustard, which contains a sulphocyanide. The filtrate from the lead meconate precipitate can be examined for morphine by removing excess of lead by means of sulphuretted hydrogen, filtering, evaporating to dryness at a low temperature, and then extracting morphine from the residue by the acetic ether and ether process (see p. 257).

Codeine

This alkaloid of opium possesses somewhat similar properties to those of morphine, but it is less powerful in its effects. Walsh [1] describes a case of poisoning by cough linctus containing codeine, in which about eight grains of the alkaloid were taken for suicidal purposes by a man suffering from phthisis. A quarter of an hour after taking the poison, emetics were administered and a quantity of the poison was vomited up. The pupils were contracted and did not react to light, the pulse was full and bounding, and the patient complained of confused feeling in the head. There was general itching of the skin, which recurred on the second day. The most noteworthy symptom was the intense irritation of the sensory nerve-endings. By the second day the patient had completely recovered.

[1] *Brit. Med. Jour.*, 1889.

CHAPTER XVI

Poisoning by strychnine—Distinction between strychnine convulsions and the convulsions of tetanus—Vermin-killers containing strychnine—Poisoning by brucine—Nux vomica.

Nux Vomica and Strychnine

Nux vomica seeds owe their poisonous properties mainly to strychnine, and to a lesser extent to brucine. Both these alkaloids are contained in the seeds of the *Strychnos Nux Vomica* (Nat. Ord. Loganiaceæ), in combination with igasuric and lactic acids. Strychnine and brucine are also contained in the seeds of *Strychnos Ignatii*, commonly called Saint Ignatius' Beans. The bark of the *Strychnos Nux Vomica* contains strychnine and brucine; it is sometimes known as 'false angustura bark.'

The smallest fatal dose of nux vomica is thirty grains, about the weight of one seed; three grains of the alcoholic extract have proved fatal. The symptoms and treatment of poisoning by nux vomica are similar to those described in connection with strychnine.

Strychnine is very slightly soluble in cold water; one part dissolves in about 8,300, but in boiling water one part dissolves in 2,500. It is much more soluble in spirit, and is very soluble in chloroform, and in a mixture of equal parts of chloroform and ether. It is a very bitter substance, one part in 70,000 of water giving a perceptibly bitter taste. Strychnine is a very stable body, and withstands prolonged contact with strong sulphuric acid at the temperature of the water-bath. If strychnine be present in viscera, it remains unchanged during the

putrefactive processes that bring about the decomposition of the viscera. Wolff found strychnine in an exhumed corpse 322 days after death. Prescott found strychnine in the stomach, liver, and intestines, in a body exhumed one year and three days after death. Allen kept in a jar for six years a portion of the stomach and liver of a person who died from strychnine poisoning; in the dry residue left in the jar at the end of that time abundance of strychnine was detected. Richter found strychnine at the end of eleven years in putrid tissues exposed for that time in open vessels.

Symptoms.—In poisoning by strychnine the symptoms appear on an average in from five to twenty minutes. This represents the usual time of commencement of symptoms if strychnine be taken in solution or in some readily absorbable form; but if taken in the solid form, such as in pills, and especially in hard and compact pills, a much longer interval may elapse before the symptoms commence. Taylor relates a case of a boy, aged twelve, who swallowed a pill containing three grains of strychnine; no symptoms appeared for three hours, at the end of which time they set in and death took place in ten minutes. The pill had been prepared with mucilage eight months previously, and, therefore, was very hard and only capable of undergoing slow solution in the stomach. In the celebrated Palmer case, Cook had administered to him two pills, presumably containing strychnine, and no symptoms were observed for an hour and a quarter. Cooke[1] relates a case of a man who had taken strychnine together with alcohol. The stomach-pump was employed half an hour later, the fluid then withdrawn emitting a strong odour of alcohol. One hour and a quarter after taking the poison the symptoms of strychnine poisoning commenced. There may be, however, a still further prolongation of the interval if a narcotic such as opium has been taken simultaneously. Macredy[2] reports a case in which a grain and a half of strychnine and two ounces of laudanum were taken. Symptoms of strychnine

[1] *The Lancet*, 1890. [2] *Ibid.* 1882.

poisoning did not appear till eight hours later. Wormley[1] mentions a case in which three grains of strychnine, one drachm of opium, and some quinine were taken together; no symptoms of strychnine poisoning appeared until twelve hours had elapsed. On the other hand, cases have been recorded in which symptoms of strychnine poisoning have occurred within four minutes from the time of swallowing the poison. Symptoms are slower in making their appearance in connection with poisoning by nux vomica.

Poisonous doses of strychnine produce violent convulsions, with spasm of the respiratory muscles, and consequent death from asphyxia and exhaustion. The convulsions are due to the irritant effect of the strychnine upon the motor centres of the cord, an enormous increase in the reflex excitability of those centres resulting from the action of the strychnine. A very slight stimulus applied to the skin is sufficient to produce the convulsions reflexly, such, for instance, as a current of air, or the touching of the patient. The strychnine spasms may also be started reflexly by a sound, such as the banging of a door, or the pouring out of water, or by the flashing of a bright light. During the convulsions, the muscles of respiration remain contracted in inspiration, and so give rise to asphyxia. The symptoms of strychnine poisoning are :—(i) A sense of uneasiness and restlessness, with a feeling of impending suffocation, which are the earliest or premonitory symptoms of strychnine poisoning. (ii) Shuddering or trembling, with twitchings and jerkings of the limbs and head. (iii) Convulsions, tetanic in their character; the spasms are at first clonic, and then for a time become tonic. The following are the peculiarities of the strychnine convulsions. (*a*) Nearly all the muscles are simultaneously affected. (*b*) The limbs are stretched out involuntarily, the hands are clenched, and the soles of the feet bent inwards, the legs being somewhat separated. (*c*) The head is bent forcibly backwards and the body becomes arched, the front of the body becoming projected, so that the person rests only

[1] *Micro-Chemistry of Poisons*, 1867.

upon the back of the head and the heels, there being a distinct space between the back of the body and the surface upon which it is resting; this position is known as *opisthotonos*. Under very exceptional circumstances the body may become arched forwards, that is in the opposite position to that of opisthotonos, or very occasionally the arching may be sideways. (*d*) The muscles of the lower jaw are generally the last affected; whereas in tetanus from disease they are amongst the first muscles affected. (*e*) Slight causes bring on the convulsions, such as a noise, a draught of air, or gently touching the patient. (*f*) There is no unconsciousness during the convulsions, and the intellect is clear; the patient suffers severely while the spasms last, and has a great apprehension of death. (iv) The eyeballs are prominent during the continuance of the spasms and the pupils are dilated; during the interval of relaxation the pupils contract. (v) During the convulsions there is cyanosis from spasm of the respiratory muscles and consequent non-aeration of the blood; after a minute or longer the spasm passes off, the respiration recommences, and the cyanosis disappears; at this period the patient is generally bathed in sweat. The intervals between the convulsive seizures last from a few seconds to five or ten minutes. (vi) Excessive thirst is experienced, but the endeavour to alleviate this by drinking frequently produces a convulsion. Vomiting may occur.

As a rule, within two hours from the commencement of the symptoms the patient either dies or recovers; if death be supervening, the convulsions rapidly succeed each other, increasing in severity, and the patient dies asphyxiated and exhausted. The mind may either remain clear to the last, with a strong apprehension of death, or shortly before death there may be a loss of consciousness. In cases of strychnine poisoning the temperature often rises after death.

The convulsions of strychnine poisoning have been mistaken for the convulsions produced by certain diseases. The convulsions of tetanus are those which are most likely to be confounded with the convulsions of strychnine poisoning, but they can be

very easily distinguished. Of the two varieties of tetanus, idiopathic and traumatic, the idiopathic is extremely rare in temperate climates, whilst in connection with traumatic tetanus a history of a previous injury may generally be obtained, such as a lacerated, punctured, or contused wound, although it should be borne in mind that a very trifling wound, such as may be inflicted by a splinter or by a rusty nail, may after a lapse of several days give rise to tetanus. The symptoms of tetanus usually commence from five to fourteen days after the infliction of the wound, but they may set in earlier or later. In one recorded case a negro lacerated his thumb by the fracture of a china dish, and was seized almost instantly with convulsions, and died with tetanic symptoms in a quarter of an hour. In the table on p. 278 will be found the main distinctions between strychnine convulsions and those due to tetanus.

The convulsions produced by strychnine have been occasionally mistaken for those of epilepsy, but there should be no difficulty whatever in distinguishing between them. The mode of seizure, the loss of consciousness, and the peculiar clonic movements of the epileptic fit, are entirely different from the convulsions of strychnine poisoning. In addition, the deep stupor occurring at the end of an epileptic attack presents a marked contrast to the complete muscular relaxation, and the clearness of intellect, following a strychnine spasm.

In the case of *Reg.* v. *Thomas Neill Cream* (C.C.C., 1892) a witness, who was an unqualified assistant to a medical man, stated in his evidence that he had formed the opinion that the symptoms, from which a young woman named Matilda Clover suffered previous to death, were those due to delirium tremens, although from the account given at the trial they were obviously caused by strychnine poisoning. It seems scarcely necessary to give any details for distinguishing between delirium tremens and the effects of strychnine. Stevenson, giving evidence for the Crown at the trial, stated 'that in delirium tremens the mental faculties are obscured, but in strychnine spasm they are very acute, and not at all impaired, except, perhaps, at the last

moment of life.' The delirium and hallucinations of delirium tremens bear no resemblance whatever to the symptoms of strychnine poisoning.

Convulsions caused by strychnine	Convulsions caused by tetanus of disease
I. The patient would be in usual state of health previous to commencement of attack ; a feeling of general uneasiness and restlessness precedes for *only a few minutes* the sudden outburst of convulsions	I. Soreness and stiffness of the muscles of the neck and jaws for several hours previous to tetanic convulsions, with a gradually increasing stiffness of the neck and difficulty in separating the jaws
II. Trismus, or lockjaw, is secondary to the spasms affecting the muscles of the limbs and trunk	II. Trismus precedes the general spasms
III. The muscles of the jaw are relaxed in the intervals between the attacks	III. Trismus remains during any mitigation of the general spasms
IV. All the muscles of the body are simultaneously and suddenly thrown into spasm, producing opisthotonos	IV. After the muscles of the jaws and neck are affected, the rigidity gradually spreads to the muscles of other parts of the body, generally to the trunk first, and then to the legs, opisthotonos being produced in the great majority of cases
V. The strychnine paroxysm lasts from half a minute to two minutes, and is succeeded by complete relaxation of the muscles until the next seizure occurs	V. The rigidity is generally permanent, or, if a remission occur, there is no complete disappearance of spasm as in the intermission of strychnine poisoning
VI. Reflex excitability is an early symptom	VI. Reflex excitability is a late symptom
VII. Epigastric pain is absent	VII. Epigastric pain (probably from spasm of the diaphragm) is severe and frequent
VIII. Death usually occurs in two or three hours from the commencement of the symptoms	VIII. Death occurs much later, rarely within twenty-four hours, usually delayed for several days

Treatment and antidotes.—If possible, the stomach should be emptied and washed out by means of the stomach-pump or tube, or an emetic of mustard and water should be given. Chloral and chloroform are the most useful antidotes that can be employed in cases of strychnine poisoning. Chloral is best administered by hypodermic injections of five grains of hydrate of chloral dissolved in ten minims of water, the injection being repeated every ten minutes until the strychnine convulsions are subdued. At the same time it is advisable to administer chloroform in the form of an inhalation to the patient, and to continue the administration of it for some time, or as long as there is any sign of the renewal of the convulsions on the removal of the chloroform. If the stomach-pump or tube be used, a drachm of bromide of potassium dissolved in three or four ounces of water may be passed into the stomach, after that organ has been washed out. Anrep recommends urethan as being superior to chloral hydrate, and advises the administration of one to one and a half drachms if a poisonous dose of strychnine has been taken.

Post-mortem appearances.—If the person has died during a spasm, the muscles may quickly enter into a condition of strong rigor mortis, which may persist for a somewhat lengthened period; but if the person has died exhausted, during a remission or interval between the spasms, rigor mortis may not be so well marked. As to the duration of rigor mortis there are many conflicting statements. In the case of Cook the body was found five days after death with rigor mortis more pronounced than is usually the case at that period, but in other cases the rigidity has been of the usual duration, and has occurred at the usual period after death. Internally there are no characteristic appearances. In strychnine poisoning there is sometimes congestion of the brain and its meninges, and of the spinal cord, and occasionally there is congestion of the lungs. The heart is contracted and empty, or the right side is distended with liquid blood. Maurel,[1] who has made a series of experimental

[1] *Bulletin de Thérapeutique*, 1892.

researches on the action of poisons on leucocytes, finds that the toxic action of strychnine on the animal economy is in the ratio of its destructive action on the leucocytes, the death of the leucocytes and that of the animal being simultaneous. The immediate effect of the poison on the white corpuscles is to arrest their spontaneous activity and fix them in the spherical state.

Fatal dose.—The smallest quantity that has caused death in an adult is half a grain of the sulphate of strychnine; a little over a grain has frequently proved fatal, so that the fatal dose of strychnine for an adult ranges from half a grain to two grains. The smallest quantity of strychnine that has ever killed is one-sixteenth of a grain, which caused the death of a child, aged two and a half years; on the other hand, recoveries have taken place after the swallowing of much larger quantities than those previously mentioned, provided the patient has been fairly promptly treated. Jones[1] records a case of recovery from strychnine poisoning, in which the quantity swallowed was estimated at six grains. Schauenstein[2] relates a case in which from seven to nine grains of strychnine nitrate were taken, and the dose followed after about half an hour by nine grains of morphine acetate; subsequently, the individual (a would-be suicide) chloroformed himself. Two hours and a quarter after taking the strychnine, convulsions occurred with well-marked opisthotonos. Emetics were employed, and tannin and codeine given separately; ultimately he made a complete recovery. Wallace and McRae[3] describe a case of strychnine poisoning, attended by recovery, after a man had swallowed twenty grains of pure strychnine sulphate. About ten minutes after taking the poison prompt vomiting was induced, the stomach-pump was used, and bromide of potassium and chloral administered. The man made a complete recovery, due no doubt to the prompt and thorough evacuation of the stomach, and also to the fact that the stomach was full of food at the

[1] *The Lancet*, 1889. [2] *Deutsche Klinik.*, 1861.
[3] *Brit. Med. Jour.*, 1892.

time the strychnine was taken. Dixon Mann[1] mentions a case in which twenty-two grains of strychnine remained in the stomach for two hours before vomiting occurred, and yet recovery took place.

Fatal period.—The most rapid death following strychnine poisoning is one in which a fatal result occurred five minutes after the commencement of symptoms, and ten minutes after swallowing the poison. As a rule death occurs, in connection with strychnine poisoning, within two hours; the presumption being that, if a person after taking strychnine survive for two hours, recovery will take place. On the other hand, the fatal period may be delayed for a few hours, as is shown by the following cases: Harley[2] reports a case of a woman who died five hours and a half after taking strychnine in the form of pills; from the vomit, stomach and contents, liver, and one kidney, Stevenson extracted 3·26 grains of strychnine. Taylor mentions the case of an adult who died in six hours from a dose of three grains of strychnine. The longest fatal period on record occurred in connection with a case described by Henry,[3] in which a man swallowed as much strychnine as would cover a shilling, probably from seven to ten grains; symptoms appeared in from ten to fifteen minutes. The stomach was subsequently washed out repeatedly, large doses of chloral and bromide of potassium given, and these were supplemented by occasional chloroform inhalations; but finally death occurred from strychnine spasms eight hours and three-quarters after the onset of the symptoms, or nine hours after taking the poison.

Elimination of strychnine.—Strychnine is eliminated in the urine, fæces, and saliva. It is rapidly removed by the kidneys; after the administration of strychnine in medicinal doses it has been found in the urine half an hour after administration; both Kratter and Dixon Mann have failed to find it in the urine forty-eight hours after its administration had ceased, indicating its rapid elimination.

[1] *Forensic Med. and Toxicol.* [2] *The Lancet*, 1893. [3] *Ibid.*

Vermin-killers containing strychnine.—Many cases of fatal poisoning by strychnine have occurred from the employment of certain vermin-killers for homicidal or suicidal purposes. Of these preparations, the one most largely in use is known as 'Battle's vermin-killer,' which contains strychnine mixed with flour or potato starch, and coloured with prussian blue. A threepenny packet of this vermin-killer contains from one to two grains of strychnine, and a sixpenny packet from two to three grains. Another preparation known as 'Butler's vermin-killer' contains strychnine mixed with flour, and coloured with soot. A sixpenny packet of this preparation contains from two to three grains of strychnine. In cases of poisoning or suspected poisoning by either of these vermin-killers the colouring matter should be carefully searched for in the contents of the stomach or intestines. In some of the vermin-killers ultramarine is used as the colouring matter, and in poisoning by such a preparation no coloured particles may be found in the stomach after death, as the acid of the gastric juice is sufficient to destroy the colour of ultramarine. Occasionally, though rarely, vermin-killers are coloured pinkish, carmine or vermilion being then employed.

Analysis and tests.—Strychnine is extracted from organic admixture and from the various viscera by the process described for the extraction of alkaloids (see p. 254). The alkaloidal residue which is obtained by the evaporation of the chloroform and ether mixture, after the first extraction from the alkaline liquid, generally contains a little colouring matter, which would interfere with the colour-test employed for strychnine. To get rid of this colouring matter the alkaloidal residue left after the evaporation of the chloroform and ether should be moistened with strong sulphuric acid and heated on a water-bath for two or three hours. In this way the organic matter is thoroughly charred and decomposed by the sulphuric acid, whereas the strychnine, which is converted into a sulphate, remains unaffected. The mixture is then diluted with water and filtered from the carbonaceous matter that has been set free; the

filtrate is rendered alkaline with ammonia, and is agitated with chloroform or the chloroform and ether mixture. The chloroform or chloroform and ether mixture is removed by a separating funnel, and if it be then allowed to fall drop by drop into a heated porcelain dish, the strychnine is obtained as a deposit in the pure state, and confined to a very small area of the dish. To the deposit so obtained in porcelain dishes the following tests may be employed.

I. Strychnine possesses a powerfully bitter taste, which is not always noticeable at once when a solution of strychnine is placed upon the tongue, but which develops quickly, and is very persistent in its effect.

II. If the solid strychnine residue be dissolved in a few drops of cold strong sulphuric acid, and an oxidising agent added, a characteristic and regular succession of colours is produced. A rich blue colour is first obtained, passing to violet, purple, and finally to cherry-red; the latter colour is persistent for some time, but ultimately changes to yellow. The following oxidising agents may be employed to produce this succession of colours, viz. manganese dioxide, lead dioxide, potassium bichromate, potassium ferricyanide, or potassium permanganate. Of these oxidising agents, manganese dioxide employed in small quantities gives the best results, the different colours being well developed and the change more gradual than with the others. To apply the test to the solution of strychnine in strong sulphuric acid contained in a porcelain dish, a small glass rod should be dipped in strong sulphuric acid, so as just to moisten it, and then a very small amount of solid manganese dioxide taken up on it; on stirring the strychnine solution with the portion of the glass rod to which the manganese dioxide is adhering, a deep blue colour is developed, changing gradually, as previously mentioned, to violet, purple and cherry-red. It should be remembered that this beautiful test for strychnine does not depend upon the production of a blue colour, but upon the regular succession or play of colours. So employed, the test is a very characteristic and delicate one, the $\frac{1}{20000}$ of a

grain of strychnine being capable of detection by it. If potassium bichromate be used, instead of manganese dioxide, the change of colour is much more rapid, and moreover the green chromium sulphate that is formed tends to mask the colours produced by the strychnine.

On account of the importance of this test for strychnine, it is advisable to consider whether any substances may interfere with the production of the colours, or whether any other substances than strychnine are liable to be mistaken for it. The presence of morphine to a certain extent interferes with the colour-test for strychnine, and if the morphine be in excess of the strychnine, then the latter alkaloid is not discoverable by the colour-test. Morphine in small quantities, however, does not interfere with the working of the test; moreover, in the extraction process for strychnine the presence of any large quantity of morphine is excluded by its very slight solubility in the chloroform and ether mixture. Other substances, however, give with strong sulphuric acid and an oxidising agent a colour somewhat resembling the strychnine colour reaction. These bodies are curarine, gelsemine, veratrine, cod liver oil, salicin, santonin, narceine, papaverine, solanine, and aniline. These substances can be easily distinguished from strychnine in that, with the exception of aniline, they are all coloured by sulphuric acid alone, either in the cold or on warming; whereas strychnine is not coloured by cold or warm sulphuric acid alone. Aniline gives no colour with sulphuric acid alone, but

Fig. 19.—Crystals of Strychnine Chromate obtained from $\frac{1}{1000}$ of a grain of Strychnine (Magnified 80 diameters)

with sulphuric acid and an oxidising agent it produces a green colour changing to blue and ultimately black, which change of colours distinguishes it from strychnine. Of the other substances mentioned, curarine and gelsemine are the only two alkaloids behaving exactly like strychnine with sulphuric acid and an oxidising agent; but, as previously mentioned, they are coloured by sulphuric acid alone. Gelsemine gives with sulphuric acid a green colour turning to red. Curarine gives with sulphuric acid a deep blue or violet colour, and with strong nitric acid it is coloured deep red or purple. Amthor[1] has described a cadaveric alkaloid or ptomaine with strychnine-like properties, which he has twice obtained from parts of a corpse eight days old. The chief differences between this alkaloid and strychnine are: (i) It is less poisonous than strychnine when injected subcutaneously into a frog. (ii) The taste is less bitter. (iii) The precipitates produced by this ptomaine with potassium chromate and picric acid are amorphous, whereas those produced by strychnine are crystalline (figs. 19 and 20). (iv) The blue colour with sulphuric acid and an oxidising agent is less persistent and less pure than the colour formed with strychnine. The formation of such a ptomaine must, however, be very rare, as the tests for strychnine have been employed by different toxicologists with negative results in an enormous number of cases, without the detection of this ptomaine; moreover, such a ptomaine would,

Fig. 20.—Crystals of Strychnine Picrate obtained from $\frac{1}{1000}$ of a grain of Strychnine (Magnified 80 diameters)

[1] *Chem. Zeit.*, 11, 288.

in all probability, be destroyed by warming with strong sulphuric acid over the water-bath.

A very delicate method of employing the oxidation test for strychnine is that suggested by Letheby, which consists in the employment of nascent oxygen, produced by electrolysis, instead of any of the oxidising agents previously mentioned. The strychnine residue is dissolved in two or three drops of strong sulphuric acid, which are placed in a small porcelain crucible. On dipping, for a moment, into the liquid two platinum wires connected respectively with the zinc and platinum plates of a Grove cell, a violet colour flashes out, and on removing the wires the tint remains.

III. Bloxam[1] has described the following characteristic and delicate test for identifying strychnine. The solid alkaloid, on a glass slide or porcelain dish, is dissolved in a drop of nitric acid and gently heated; to the warm solution a very minute quantity of powdered potassium chlorate is added, which produces an intense scarlet colour. The addition of one or two drops of ammonia changes this colour to a brown, and a brownish precipitate falls. If the mixture be then evaporated to dryness, it leaves a dark green residue, which, dissolved in a drop of water, gives a green solution, changed to orange-brown by caustic potash, and becoming green again with nitric acid. No other of the commonly-occurring alkaloids can be mistaken for strychnine by this test.

IV. The physiological test for strychnine is carried out by injecting a few drops of a solution of strychnine under the skin of the back of a small frog; the animal is then placed under a glass shade and watched. Tetanic convulsions generally occur in a few minutes, even if only a minute quantity of the strychnine be present. If convulsions have once occurred, they can be reproduced by either touching the frog, or by smartly tapping the table upon which it is resting.

V. Potassium bichromate produces with a solution of a strychnine salt a bright yellow precipitate of the chromate of

[1] *Chem. News*, lv. 155.

strychnine, which almost immediately becomes crystalline. This reaction can be carried out on a microscope slide, and the shape of the crystals observed under the microscope (fig. 19). If the crystals are allowed to dry, and are then touched with a small drop of strong sulphuric acid, a deep blue colour is obtained, and quickly changes to violet, purple, cherry-red, and ultimately to green; so that the crystalline precipitate of strychnine chromate enables the oxidation colour-test to be applied by way of confirmation.

VI. Picric acid gives with solutions of strychnine salts a yellow precipitate of strychnine picrate (fig. 20).

Failure to detect strychnine.—Occasionally, careful analysis has failed to detect this poison after death. Reese mentions a case that occurred to him where a woman was alleged to have been poisoned by six grains of strychnine, and in whose case death was postponed for the long period of six hours. The body was examined by him for poison eight weeks after death, and although the body was well preserved, he failed to detect strychnine. In this case morphine had also been administered, and might possibly have interfered with the colour-test. If a small, but fatal, dose of strychnine has been taken or administered, and if death does not occur until more than two hours have elapsed, it is possible that so much of the strychnine may have been eliminated, and the remaining minute amount be so widely distributed throughout the body as to escape detection; so that the non-detection of strychnine does not prove that it has not destroyed life. If, however, death occur within a couple of hours of the taking or administration of strychnine, Stevenson, who has had a considerable experience in the detection of this poison, is of opinion that it can hardly fail to be detected in the body.

Brucine

Brucine is an alkaloid that is found along with strychnine in nux vomica seeds, and in the Ignatius bean. As it is an

alkaloid that is practically unknown to the public, cases of poisoning by it seldom occur. Physiologically it considerably resembles strychnine, but is less poisonous; the physiological activity of strychnine is stated by different observers to be thirty-eight, twenty-four, and ten times greater than that of brucine. Brunton [1] finds that brucine acts in a very similar manner to strychnine, and produces death by convulsions after injection; when taken into the stomach, he finds that it is rapidly eliminated in the urine, and does not then so frequently produce convulsions. The symptoms and treatment are the same as those given in connection with strychnine poisoning.

Tests.—I. The most delicate test for brucine is the nitric acid reaction. If to a solid brucine residue a drop or two of strong nitric acid be added in the cold, a bright arterial blood-red colour is produced, which, on warming, changes to yellowish-red and yellow. If the mixture be now cooled, and stannous chloride very cautiously added, a purple colouration is produced, which is destroyed by excess of the stannous chloride. If, after the addition of cold nitric acid to solid brucine, the red-coloured liquid obtained be largely diluted with water, a yellow precipitate separates; this precipitate is soluble in dilute hydrochloric acid. If the filtrate from the yellow precipitate be neutralised by ammonia, and tested with calcium chloride, a precipitate of calcium oxalate falls, insoluble in acetic acid, but soluble in hydrochloric acid. This last-mentioned combined test is peculiar to brucine.

II. With sulphuric acid and potassium bichromate, brucine gives a deep orange-red colour.

III. With sulphomolybdic acid solid brucine gives an orange-red or purple-red colour, which changes to greenish or blue.

IV. Blyth [2] considers that the best test for brucine is the reaction with methyl iodide. If to a solution of brucine in

[1] *Chem. Soc. Jour.*, 1885. [2] *Poisons*, 1884.

strong alcohol a little methyl iodide be added, circular rosettes of crystals appear at the end of a few minutes. A solution of strychnine gives with methyl iodide no similar reaction.

Nux Vomica

Nux vomica seeds and their preparations, such as the extract and tincture, produce poisonous effects similar to those of strychnine. The symptoms of poisoning by nux vomica are usually longer in appearing than when strychnine is taken. As previously mentioned, thirty grains of the powdered seeds, and three grains of the extract, have proved fatal. Stevenson [1] records the case of a boy aged twelve, who, after taking about eight grains of the extract of nux vomica, recovered; both strychnine and brucine were detected in the urine.

[1] *Guy's Hospital Repts.*, 1868.

CHAPTER XVII

Poisoning by belladonna (deadly nightshade) and atropine—Hyoscyamus (henbane)—Stramonium (thorn-apple)—Duboisine—Solanum dulcamara (bitter sweet)—Aconite (monkshood) and aconitine—Veratrum and veratrine—Black hellebore.

Belladonna, Hyoscyamus, Stramonium, Solanum and Duboisia

All these poisonous plants owe their active properties to alkaloids. They are all members of the natural order Solanaceæ, and are neurotic poisons, frequently known as deliriants on account of the active delirium that constitutes one of the principal symptoms when taken in poisonous doses. They are also known as mydriatics, on account of their physiological property of dilating the pupil. In general, they produce other effects in common, such as arresting the secretions of saliva and sweat, and so causing dryness of the mouth and a dry flushed skin. They frequently produce a red rash upon the skin, and visual hallucinations or spectral illusions are common in poisoning with members of this group. The active alkaloid of belladonna is atropine, of hyoscyamus—hyoscyamine, of duboisia—duboisine. All these alkaloids are isomeric—that is, their chemical composition is the same, all of them possessing the formula $C_{17}H_{23}NO_3$. Daturine, the active alkaloidal principle of the stramonium plant, is now known to be a mixture of atropine and hyoscyamine, and not a distinct alkaloid.

Belladonna and Atropine

Atropa belladonna (Nat. Ord. Solanaceæ, or Atropaceæ as it was formerly called) is a plant (Plate II.) that grows wild

Plate II

Belladonna or Deadly Nightshade (Atropa Belladonna)

in this country; it is commonly known as the *deadly nightshade*. The berries, leaves, and root are poisonous, their poisonous properties being due to the alkaloid atropine. The berries when ripe possess a dark purple colour with a bloom upon them, and are very attractive in appearance to children and others, by whom they are not unfrequently eaten, with the consequent production of belladonna poisoning. Each berry contains several dark-coloured seeds (fig. 21). In addition, the Pharmacopœial preparations of belladonna occasionally give rise to cases of poisoning; of these the principal ones are the tincture, liniment, and extract. The latter is frequently prescribed for external use, rubbed up with an equal quantity of glycerine, and when so prepared, it forms a viscid liquid very much resembling treacle in appearance and consistency, and may be taken by children for such by mistake. The elimination of atropine takes place rapidly by the kidneys.

FIG. 21.—BELLADONNA SEEDS
A, natural size; B, magnified 6 diameters

Symptoms.—The symptoms of belladonna or atropine poisoning are similar to those produced by other members of this group of neurotic and deliriant poisons, so that the following description of the symptoms will suffice for the other members of the group. (i) A hot and dry sensation in the mouth and throat, accompanied by thirst, and followed by paralysis of the muscles of deglutition, swallowing being difficult or impossible in consequence. (ii) Nausea and vomiting sometimes occur, especially if portions of the plant have been eaten. (iii) Dilatation of the pupils and indistinctness of vision, owing to paralysis of the sphincter iridis and the ciliary muscle. Dilatation of the pupils is well marked, the iris frequently being reduced to a thin ring. The indistinctness of vision is in part due to paralysis of the power of accommodation, and in part,

no doubt, to the action of the poison on the brain centres. (iv) The face is flushed, the eyes are sparkling, and the pulse is small and, as a rule, very rapid. (v) Giddiness quickly supervenes, followed by restlessness, great excitement, and frequently by delirium; the delirium is generally attended with visual hallucinations and spectral illusions, and is frequently imitative in its type, such movements as those of a person employed in sewing, or of one cutting cloth into strips, or picking substances, being imitated and persisted in for a considerable time. (vi) Drowsiness and stupor generally supervene. In the graver cases convulsions also may occur. (vii) In the greater number of cases an erythematous rash, resembling the rash of scarlet fever, occurs upon the chest and other parts of the body; the temperature is also sometimes raised, but not as a rule above 101° or 102°. On account of the conjunction of the flushed face, the delirium, the scarlatinal-like rash, and the elevation of temperature, a case of belladonna poisoning may be mistaken for a case of scarlet fever, if the history of the attack be not taken into account. Recovery is slow, and constipation during recovery is not uncommon.

Fairly typical symptoms of poisoning have occasionally occurred after the dropping or instillation of a solution of atropine into the eyes for ophthalmic purposes. Owens[1] records two such cases, one in which one-twelfth to one-eighth of a grain of atropine was instilled into the eyes of a boy, aged twelve, in ten instillations spread over two days; after the last instillation he showed most of the symptoms of atropine poisoning—viz. A staggering and unsteady gait, dryness of the throat and tongue, flushed face, pupils moderately dilated, picking at imaginary objects in the air, incoherent muttering, smiling, and occasionally laughing outright to himself. He could be roused to attention for a few minutes, but soon relapsed. A few hours later tetanic spasms came on with delirium. Complete recovery took place after a few days. The second case was that of a healthy male adult, aged seventy-three, into whose eyes one

[1] *The Lancet*, 1890.

twenty-fifth of a grain of atropine was instilled in three instillations extending over twenty minutes. The pupils were dilated, and shortly afterwards the patient became almost unconscious; his face was suffused, the body covered with an erythematous rash, the pulse was extremely rapid, and there was almost complete paralysis. Recovery in this case took place in a few days. Purley[1] relates the particulars of a case in which about two-fifteenths of a grain of homatropine were instilled into the eyes of a child, aged seven, by four instillations extending over a period of an hour. Shortly afterwards the child became restless and excited, the face was flushed, the expression of her ideas became very incoherent and extravagant, and she saw imaginary objects. The gait became staggering, and mild delirium set in. Complete recovery occurred after some days.

Symptoms of belladonna poisoning may also occur from the external application of a belladonna plaster, if the surface of the skin to which it is applied become irritated, inflamed, or blistered. Walker[2] describes a case of a female, aged sixty-five, who applied a perforated belladonna plaster about eight inches square to her back. Ten hours after its application the skin became inflamed and vesicated, and the plaster was removed. At the end of three weeks the belladonna plaster was again applied, and seven hours afterwards symptoms of poisoning set in. They consisted of giddiness and staggering gait, delirium followed by complete loss of power, unconsciousness, rigidity of limbs, with convulsive twitchings, wide dilatation of pupils, and retention of urine. On removing the plaster the skin below it presented the appearance of a healing blister. Morphine was injected hypodermically, and in twenty-four hours the patient was convalescent. Howarth[3] describes a case of rapid supervention of symptoms of poisoning following the application of a belladonna plaster to the lumbar region. A man, aged fifty-seven, applied a belladonna plaster about six inches by four to the lumbar region, which had been ren-

[1] *New York Med. News*, 1892.
[2] *Brit. Med. Jour.*, 1891. [3] *The Lancet*, 1894.

dered hyperæmic by previous friction with mustard oil. After about three-quarters of an hour, dryness of the mouth and tongue supervened, followed by numbness of the hands and feet, and shortly afterwards by dimness of vision and confusion of mind. About one hour and a quarter after applying the plaster vomiting came on, and was followed by unconsciousness and delirium, which was incoherent and of the muttering amused type, and he frequently burst out laughing in a hearty manner. On removing the plaster no abrasion of the skin was found underneath, but it was markedly hyperæmic from the application of the stimulating oil; there was retention of urine. Under treatment with the extract of physostigma, rapid recovery took place. Several cases of severe poisoning have also resulted from the application of atropine ointment to ulcers.

Treatment and antidotes.—The stomach should be emptied and thoroughly washed out by means of the stomach-pump or stomach-tube, or, if neither of these be procurable, an emetic of mustard and water should be given. The best and most rapidly acting antidote to atropine is pilocarpine. One-third to one-half of a grain of the nitrate of pilocarpine should be hypodermically injected, and the injection, if necessary, should be repeated an hour later, the indications for this repetition being the continued dilatation of the pupils and dryness of the skin. The beneficial action of pilocarpine is indicated by commencing contraction of the pupils, sweating, and abatement of the delirium and other symptoms. Stimulants and hot coffee should be administered. In the collapse stage artificial respiration may be found necessary. The administration of morphine hypodermically in cases of poisoning with belladonna or atropine has been frequently recommended, and certainly the tolerance of morphine exhibited in atropine poisoning is striking, and is in favour of the antagonism of these drugs; but, although atropine may be of use in cases of opium or morphine poisoning, the converse is not by any means so certain. If pilocarpine be not at hand, hypodermic injections of morphine may be employed in cases of belladonna or atropine

poisoning, but it should only be used in the stage of excitement, and not in the final stage of collapse, when its use would be dangerous. An interesting example of the good effects of pilocarpine is related by Campbell,[1] in which a male, aged sixty, had administered to him by mistake a tablespoonful of a liniment consisting of equal parts of belladonna liniment and tincture of opium. Although the so-called physiological antidote, morphine, was present in this liniment, the typical symptoms of belladonna poisoning supervened. Recovery took place after hypodermic injections of pilocarpine, which were repeated until one grain of pilocarpine in all was injected ; the patient lapsed into a comatose condition during an interval of about six hours, whilst a fresh supply of pilocarpine was being obtained.

Post-mortem appearances.—There are no characteristic appearances of belladonna or atropine poisoning. Careful examination of the contents of the stomach and intestines should be made for portions of berries and seeds. The mucous membrane of the stomach may be found stained of a purplish colour with the juice of the berries, if poisoning has resulted from their use. Maurel,[2] who has made a series of experimental researches on the action of poisons on leucocytes, finds that the toxic action of atropine is in the ratio of its destructive action on the leucocytes. He discovered the interesting fact that in the case of the hare—an animal which eats belladonna with impunity—atropine was almost without effect on the leucocytes of that animal. From his experiments, Maurel considers it probable that the leucocytes have a principal part to perform in poisoning by atropine.

Fatal dose.—One teaspoonful of belladonna liniment and one drachm of the extract of belladonna respectively have proved fatal. McGowan [3] relates a case of recovery after a tablespoonful of belladonna liniment had been taken, and Oliver [4] relates a case of recovery after a patient had swallowed a mixture of equal

[1] *Brit. Med. Jour.*, 1893.
[2] *Bulletin de Thérapeutique*, 1892.
[3] *The Lancet*, 1890.
[4] *Ibid.*, 1891.

parts of the extract of belladonna and glycerine (containing about half an ounce of the extract of belladonna). In both these cases pilocarpine was used as an antidote. Fourteen belladonna berries caused the death of an old man, but recovery has taken place after eating fifty berries. Children are not so susceptible to the poisonous action of belladonna as adults; recovery has followed in a child after eating thirty berries.

Of atropine, half a grain is the smallest known fatal dose for an adult. Greenway[1] records the case of a man, aged forty-five, who swallowed, by mistake, a teaspoonful of solution of atropine sulphate containing half a grain of the sulphate; symptoms of poisoning followed, but death did not occur until the sixth day. Two grains of atropine have also proved fatal in an adult. Recovery has taken place after taking five grains and a half of atropine sulphate; and a child, two years and a half old, has recovered from a quarter of a grain of atropine.

Fatal period.—The shortest period within which death has resulted from the taking of atropine is twelve hours. Usually death occurs within twenty-four hours, but it may be delayed much later, as in the remarkable case described above in which death did not occur until the sixth day.

Analysis and tests.—Careful examination should be made of the contents of the stomach and intestines for berries and seeds as previously mentioned. The mucous membrane of the stomach may be stained of a purplish colour from the colouring matter of the berries; this colouring matter has a deep purple hue, and is turned green by alkalies and red by acids. For the extraction of atropine from the contents of the stomach or intestines, or from the viscera, the process described on p. 254 should be employed. The chemical tests are not alone conclusive, but should be used in conjunction with the physiological test. At the same time it should be borne in mind that the chemical and physiological tests are given alike by all the tropines—viz. those derived from belladonna, hyoscyamus and stramonium—so that the identification of the group is all that

[1] *Brit. Med. Jour.*, 1878

can be done with certainty by the toxicologist, although the possible distinction of atropine from the other tropines may be made by one or two of the tests detailed below.

I. *Physiological test*—The residue left by the evaporation of the chloroform and ether mixture, at the end of the extraction process, should first be identified as an alkaloid by the usual group reagents, and then a drop or two of a neutral aqueous solution of it should be introduced into the eye of a kitten or cat. If atropine be present, dilatation of the pupil occurs in from a few minutes to an hour, according to the amount present. A drop or two of a solution containing one part of atropine in 130,000 parts of water is sufficient to produce this effect. The test may be repeated on the eye of a human being.

II. *Vitali's test.*—To a small amount of solid atropine residue a drop or two of strong nitric acid is added, and the liquid evaporated to dryness over a water-bath. When cold, the residue is touched with a glass rod which has been dipped into a freshly prepared solution of caustic potash in absolute alcohol; a beautiful violet colouration is instantly produced, which slowly changes to dark red. This violet reaction is peculiar to atropine and its isomers—*i.e.* to the tropines. Beckmann, however, has pointed out that a somewhat similar reaction to atropine is given by veratrine with Vitali's test, but if nitrous acid or a nitrite be used instead of nitric acid, and aqueous solution of potash instead of alcoholic potash employed, atropine gives a violet-red colour, while veratrine gives a yellow colour.

III. A modification of Vitali's test is to add to a minute quantity of solid atropine a drop or two of strong cold sulphuric acid, and then a fragment of sodium nitrite. A yellow colour is produced, which, on the addition of a solution of caustic potash in absolute alcohol, changes to reddish-violet and then to pale rose.

IV. *Gerrard's test.*—One-tenth of a grain of atropine is placed on a watch-glass, and twenty minims of a two per cent.

solution of mercuric chloride in alcohol of fifty per cent. strength gradually added; a red colouration is produced at once by atropine. Hyoscyamine gives under similar conditions a yellow colour, and, on heating, a red precipitate is formed. Hyoscine gives neither a red nor yellow colouration or precipitate. Most of the other common alkaloids give white precipitates when similarly treated. This test is only applicable in the absence of alkalies.

V. Free atropine gives a red colour with phenolphthalein. This reaction is common to the other tropines, viz. hyoscyamine, hyoscine, and homatropine, but it is not given by any other fixed alkaloid in common use, hence its value. To obtain this reaction a minute quantity of the alkaloid should be placed on phenolphthalein paper, and the fragment should then be wetted with absolute alcohol. No colouration is produced with an alcoholic solution of atropine, but on allowing the alcohol to evaporate, and touching the residue on the paper with a drop of water, a red colour appears. This colour disappears on the addition of alcohol, but is reproduced when the spirit has evaporated. This reaction is characteristic, as the caustic alkalies react with phenolphthalein in alcoholic solution.

FIG. 22.—CRYSTALS OBTAINED BY ADDITION OF A SOLUTION OF BROMINE IN HYDROBROMIC ACID TO A SOLUTION CONTAINING $\frac{1}{100}$ OF A GRAIN OF ATROPINE (Magnified 340 diameters)

VI. Blyth[1] states that a delicate test for atropine is to evaporate a speck of the alkaloid to dryness with a few drops of strong solution of baryta, and to heat the residue, when an

[1] *Poisons*, 1884.

intense odour is given off, which is analogous to that of hawthorn-blossom, and is unmistakably agreeable.

VII. Wormley[1] states that an aqueous solution of hydrobromic acid, saturated with free bromine, produces in solutions of atropine, even if very dilute, a yellow amorphous precipitate, which in a short time becomes crystalline; the precipitate may redissolve if the alkaloidal solution be strong, but is reproduced on the further addition of the bromine reagent. The production of the crystals is stated to be quite characteristic of the alkaloid (fig. 22), as all other common alkaloids produce under similar conditions a precipitate which remains amorphous. An alcoholic solution of bromine may be employed as a reagent in place of solution of hydrobromic acid.

Hyoscyamus

Hyoscyamus Niger (Nat. Ord. Solanaceæ) is commonly known as *henbane* (Plate III.) All parts of the plant are poisonous. The poisonous properties are due to two alkaloids, hyoscyamine and hyoscine; hyoscyamine is convertible into atropine. Henbane seeds (fig. 23) have been sold in mistake for celery seeds. Martin[2] describes the poisoning of four people after partaking of soup flavoured with henbane seeds, which had been sold by mistake for celery seeds; all the cases recovered under treatment. Henbane seeds are used as a popular remedy, among the lower classes, for the relief of toothache. The method of employing them is to place some red-hot coal upon a shovel or plate, and sprinkle over the

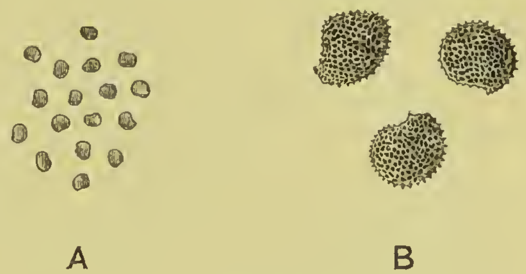


Fig. 23.—Hyoscyamus (Henbane) Seeds
A, natural size; B, magnified 6 diameters

[1] *Micro-Chemistry of Poisons*, 1867.
[2] *Brit. Med. Jour.*, 1892.

burning coal the henbane seeds, the open mouth of the sufferer being held over the coals. Under the influence of the heat the seeds sprout or germinate, and the protrusion of the thread-like radicle of the seed is taken for a worm which is supposed to have dropped out of the diseased tooth.

Symptoms.—The symptoms of poisoning by henbane are very similar to those of belladonna poisoning (see p. 291). The tendency to noisy delirium, especially with hyoscine, is not so great as with atropine; but as a rule there is more collapse and tendency to insensibility with henbane than with belladonna poisoning. Dodd[1] relates a case of poisoning which occurred in a female, aged fifty, from swallowing by mistake six drachms of the tincture of hyoscyamus (B.P.). Free vomiting was produced a few minutes after taking the poison. Twenty to twenty-five minutes later the pupils began to dilate, the pulse was 120, there was great weakness and trembling of the limbs, marked dryness of the throat, flushing of the face, the extremities became cold, and in a short time the pupils were fully dilated. Two hours after taking the poison the patient was powerless to move her legs, and the upper extremities were almost completely paralysed; slight delirium was present. Under treatment she recovered completely.

Treatment and antidotes.—The same as for belladonna poisoning (see p. 294).

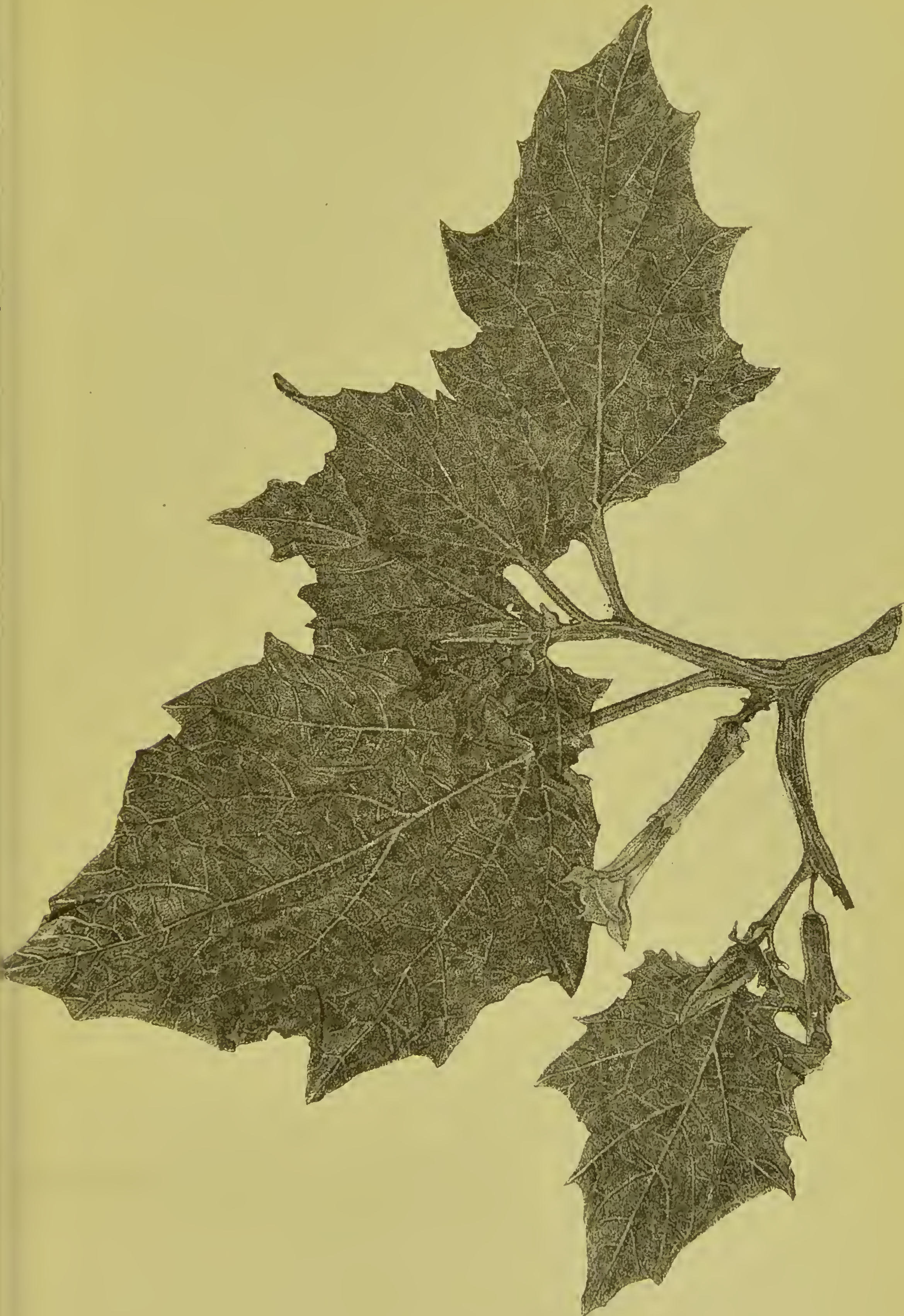
Post-mortem appearances.—Nothing characteristic.

Fatal dose.—Of henbane itself nothing is known; as in the case mentioned above, recovery has taken place after swallowing six drachms of the tincture. Death has been produced by taking one-eighth of a grain of hyoscyamine with some quantity of morphine sulphate; one-fortieth of a grain of hyoscine has produced severe symptoms.

Fatal period.—Nothing definite is known.

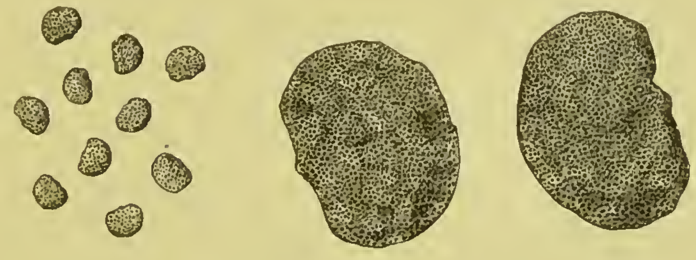
Analysis and tests.—The alkaloids of henbane may be extracted by the general process for the extraction of alkaloids (see p. 254). The tests described for atropine should then be applied to them (see pp. 296–299).

[1] *Brit. Med. Jour.*, 1889.



Stramonium

Datura Stramonium (Nat. Ord. Solanaceæ) is commonly known as *thorn-apple* and *devil's-apple* (Plate IV.) The fruit contains several dark-coloured seeds (fig. 24). It is a poison, and owes its active properties to daturine, which, however, has been shown by Ladenburg to be a mixture of hyoscyamine and atropine. In India two species of stramonium, *Datura Fastuosa* and *Datura Alba*, are largely used by the Hindoos, under the name of *dhatoora*, for criminal purposes. It is used there for the purpose of murder, or for stupefying a victim, so that he may be easily robbed or rendered for a time incapable of exercising his mental faculties. For a full account of the use of



A B

Fig. 24.—Stramonium Seeds
A, natural size ; B, magnified 6 diameters

this poison by the Hindoos, see Chevers, 'Manual of Medical Jurisprudence for India.'

Symptoms.—Similar to those of belladonna poisoning (see p. 291).

Treatment and antidotes.—The same as for belladonna poisoning (see p. 294).

Post-mortem appearances.—Nothing characteristic.

Fatal dose.—About 100 seeds, and seventeen or eighteen grains of the extract[1] have caused death.

Fatal period.—Death has occurred in seven hours.

[1] Dixon Mann's *Forensic Med. and Toxicol.*

Analysis and tests.—The usual process for alkaloidal extraction (see p. 254) should be employed. The same tests as for atropine may be used (see pp. 296–299).

Duboisine

This alkaloid is derived from *Duboisia Myoporoides* (Nat. Ord. Solanaceæ). Duboisine is an isomer of atropine, and is believed to be either identical with hyoscyamine or with a mixture of hyoscyamine and hyoscine. Duboisine produces similar symptoms of poisoning to those of atropine, and like atropine it is used as a mydriatic. Chadwick [1] relates a case of poisoning by duboisine, which occurred in a man aged seventy-five, into whose eyes two discs each containing $\frac{1}{200}$ of a grain of sulphate of duboisine were placed for ophthalmic purposes. Shortly afterwards the patient complained of giddiness, in about twenty minutes the pupils were dilated, and a few minutes later weakness, loss of control over the legs, and great dryness of the mouth and gums supervened. The patient walked, talked, and behaved like one slightly intoxicated. There was inability to recognise the position of objects, partly due to paralysis of accommodation and partly due to visual hallucinations. There was incessant activity, accompanied by a flow of words, the sentences being strung together with no apparent connection. With reference to these marked symptoms being produced by so small a quantity as $\frac{1}{100}$ of a grain, Chadwick puts the question whether advancing years increased the susceptibility of the action of the poison, as it is known that children bear belladonna better than adults. Kolloch [2] relates a very similar case caused by placing two drops of a solution of sulphate of duboisine (four grains to the ounce) in the eyes.

Treatment.—The same as for belladonna poisoning.

Solanum

Two species of solanum grow in this country, both belonging to the Nat. Ord. Solanaceæ. The commoner one is *Solanum*

[1] *Brit. Med. Jour.*, 1887. [2] *Med. News*, 1887.

Plate V.

Bittersweet or Woody Nightshade (Solanum Dulcamara)

Danielsson & Co.

Dulcamara (Plate V.), commonly known as *bitter-sweet* or *woody nightshade*, which bears small purple flowers and red berries when ripe. The other is the *Solanum Nigrum*, or *garden nightshade*, which bears white flowers and black berries. Solanine, a poisonous alkaloid, occurs in both; poisoning generally occurs from eating the berries. The berries of the solanum dulcamara, or woody nightshade, look like red currants when ripe, and, as the plant is a very common one in hedges in England, these berries are not unfrequently eaten by children; fortunately, they are not by any means so poisonous as the berries of the Atropa Belladonna, or deadly nightshade.

Symptoms.—The following symptoms occur from eating berries of either species of solanum:—(i) Pain in the stomach, headache, and giddiness. (ii) Vomiting and diarrhœa, with tenesmus and colic. (iii) Dilatation of the pupils, pallor, coldness of the surface, and thirst. (iv) Hallucinations, convulsions and coma may occur. If death occur, it is generally from asphyxia, due to paralysis of the respiratory centres.

Treatment and antidotes.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube. Warmth should be applied externally, and stimulants administered. Opium or morphine in small doses should be given.

Post-mortem appearances.—Nothing characteristic.

Fatal dose.—Nothing is known. Taylor mentions the case of a boy, aged four, who died in about twenty-four hours after eating the red berries of the solanum dulcamara, or woody nightshade.

Aconite and Aconitine

Several species of aconite exist. The plant from which the Pharmacopœial preparations should be made is the *Aconitum Napellus* (Nat. Ord. Ranunculaceæ), which is commonly grown in gardens, and which is known as *monkshood* (Plate VI.), and sometimes as *wolfsbane* or *blue rocket*. All parts of the plant are poisonous, but the greater number of deaths from aconite

poisoning have been due to eating aconite root, or to some of the medicinal preparations of aconite. The B.P. liniment of aconite is a strong preparation, about eight times as strong as the tincture. A non-official aconite preparation known as Fleming's tincture of aconite is from five to six times the strength of the B.P. tincture. Aconite root has not unfrequently been mistaken for horse-radish root, as the plant is commonly grown in gardens, and the root may be dug up in mistake for horse-radish; consequently the scraped aconite root has been served up at table in mistake for the latter. The distinction of the two is, however, very easy. Aconite root is short, tap or conical-shaped, generally from two to three inches long; horse-radish root is long, and cylindrical-shaped, generally several inches in length. Aconite root is externally of a dark coffee-brown colour; horse-radish root has a pale yellow colour. Fresh aconite root, when scraped, merely evolves a faint earthy odour; horse-radish root yields a characteristic pungent odour. Scraped fresh aconite root, after exposure to the air, turns a pinkish colour; scraped horse-radish root does not change colour. Aconite root has also been eaten in mistake for celery; a fatal case of aconite poisoning occurred recently in Norfolk from such a mistake being made.

The aconite alkaloids have been investigated by Groves, Wright, the author, Dunstan, Ince, and others. The principal alkaloid is aconitine, which is an extremely powerful poison, probably the most active poison that is known. It possesses, even in very minute amounts, a peculiar action on the tongue, lips, and throat; it produces, when a small quantity of its solution is placed upon the tongue, a peculiar tingling feeling, followed later by a numbness of the tongue, lips, and throat. This is succeeded by a sense of constriction or swelling in the throat, as if the individual were suffering from sore throat. If aconite or aconitine be taken in poisonous quantities, a similar general tingling, followed by paralysis of the sensory nerve-endings, occurs all over the body, due to the poison at first

Plate VI.

Monkshood or Aconite (Aconitum Napellus)

Danielsson & Co. sculp.

stimulating and afterwards paralysing the nerve-endings, and later the nerve-centres. Respiration is slowed, owing to the action of the poison on the respiratory centres. Death is usually due to arrest of respiration, but may be due to failure of the heart, or to both combined. Aconitine is eliminated in the urine and in the fæces.

Symptoms.—(i) Tingling, followed by numbness of the lips, mouth, and throat, accompanied by a sensation of constriction or soreness of the throat (these effects are due to the local action of the poison). (ii) Nausea, epigastric pain, vomiting and diarrhœa occasionally. (iii) Tingling, followed by numbness all over the body (due to the effect of the absorbed poison), and followed later by muscular twitchings, cramps in the arms and legs, and muscular prostration. (iv) Feeble and intermittent or irregular pulse; respirations laboured and irregular; pupils frequently dilated, but sometimes contracting at intervals. (v) Fall of body temperature, with coldness of the surface and a clammy skin. (vi) Delirium, or a tendency to drowsiness or stupor, sometimes accompanied by actual loss of consciousness; convulsions may occur towards the end. As previously mentioned, death is usually due to arrest of respiration, but may be due to failure of the heart, or to both combined. Valentine[1] describes a case of aconitine poisoning in a male adult, accompanied by somewhat exceptional symptoms. One-fourteenth of a grain of aconitine was taken; the symptoms consisted of paraplegia, stertorous and irregular breathing, the respirations varying from six to thirteen per minute, irregular pulse, and later on coma. Under treatment the patient recovered.

Treatment and antidotes.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube. If neither of these be procurable, an emetic should be employed; stimulants should be freely given; warmth and friction applied if necessary, and artificial respiration should be resorted to. Probably the best antidote to aconite is digitalis,

[1] *New York Med. Jour.*, 1888.

which overcomes the extremely depressing action of the aconitine on the heart. It should be administered by hypodermic injection of ten to twenty minims of tincture of digitalis, or $\frac{1}{100}$ of a grain of digitalin, and the dose should be repeated in twenty minutes if necessary. Robinson[1] reports the case of a soldier who took two drachms of tincture of aconite. An hour later he was extremely collapsed, and at times lapsed into unconsciousness. The stomach was emptied, and twenty-five minims of tincture of digitalis given by hypodermic injections with forty-five minims of sal volatile and two drachms of brandy; recovery took place in about four hours. Hypodermic injections of strychnine may be of use, but strychnine is inferior in its utility to digitalis.

Post-mortem appearances.—Nothing very characteristic is found in cases of aconite poisoning; congestion of the mucous membrane of the stomach and small intestines has been noticed. In the case of Percy Malcolm John, who was poisoned by Lamson with aconitine, the cardiac end of the stomach was red and inflamed, and presented a blistered appearance. There was also great congestion of the duodenum and patches of congestion in other portions of the intestines in a lesser degree. The membranes of the brain and spinal cord were congested, and congestion was also found in the lungs, spleen, liver and kidneys.

Fatal dose.—Fatal results have occurred from the taking of sixty grains of aconite root, two grains of the B.P. extract of aconite, and one drachm of the B.P. tincture respectively. *Fleming's tincture of aconite* is six times as strong as the B.P. tincture, and twenty-five minims of it have proved fatal to an adult. Crystalline aconitine is an extremely powerful poison; one-fifteenth of a grain of aconitine nitrate has proved fatal to an adult man in five hours, and Pereira mentions a case in which one-fiftieth of a grain nearly proved fatal to an elderly lady. The case of *Reg.* v. *Lamson* (C.C.C. 1882) was a trial for aconitine poisoning. Lamson, who was a medical practitioner,

[1] *Boston Med. and Surg. Jour.*, 1892.

was tried for the murder of his brother-in-law, Percy Malcolm John, by administering aconitine. For this crime he was found guilty and was executed. On December 3rd, 1881, Lamson visited his brother-in-law, a youth of nineteen, and administered to him, in a gelatine capsule, probably about two grains of Morson's aconitine, which he pretended at the time was sugar. Death occurred four hours and five minutes after administration of the poison, and about four hours after the commencement of symptoms. From some of the vomited matter, from the viscera, and from the urine taken from the bladder after death, Stevenson and Dupré extracted aconitine, and obtained with it the usual physiological tests for that alkaloid. Smith [1] relates a case of recovery after a teaspoonful of Fleming's tincture of aconite had been taken on an empty stomach by a woman aged forty. Vomiting was induced within five minutes of taking the poison, and belladonna and stimulants were subsequently administered. The symptoms consisted of a burning sensation in the præcordial and epigastric regions, a feeling of suffocation, numbness of the face and hands, pallor of the face, coldness of the surface, and irregular pulse. In about twelve hours all the symptoms had passed off. Hardmann [2] relates a case of recovery in a lady, aged twenty-four, who took by mistake thirty or forty drops of the B.P. liniment of aconite. Free vomiting was induced and stimulants employed; recovery took place in about twelve hours.

Fatal period.—Death generally occurs in from two to six hours, but it may be delayed for twenty-four hours.

Analysis and tests.—Aconitine can be extracted from vomited matters, the contents of the stomach, or from the viscera, by the general process for the extraction of alkaloids (see p. 254). Care must be taken, however, that the aconitine is not left for any length of time in contact with alkalies, as it speedily undergoes decomposition under such conditions. Stevenson finds that aconitine cannot be detected in the viscera, in which it was known to exist, after they have remained alkaline for some

[1] *Brit. Med. Jour.*, 1893. [2] *Ibid.*

time from putrefactive decomposition. There are no reliable chemical tests for aconitine, the colour reactions that have been described as occurring with phosphoric and sulphuric acids being due to impurities in the aconitine experimented with; pure aconitine does not give such colour reactions. The tests to rely on are the peculiar and extremely characteristic sensations imparted to the tongue, lips, and fauces, by minute quantities of aconitine, and experiments on mice.

I. *The taste test.*—The alkaloidal residue, obtained by the process for extraction of alkaloids, after the final evaporation of the chloroform and ether mixture should be dissolved in a few drops of water acidulated with acetic acid. The solution, if necessary, may be further diluted, according to the amount of residue originally left. A drop of the solution should then be placed on the tip of the tongue. If aconitine be present, a peculiar tingling sensation of the tongue and lips is produced in a few minutes, followed by a numbness, and if some of the liquid has passed along the surface of the tongue to the fauces, a sensation of sore throat or constriction of the fauces is felt. This effect constitutes a very characteristic and delicate test for the presence of aconitine, and is produced by a single drop of the B.P. tincture of aconite and by less than $\frac{1}{1000}$ of a grain of the alkaloid. Stevenson states that $\frac{1}{2000}$ of a grain of English aconitine may be recognised by this test.

II. *The mouse test.*—Two or three drops of the solution of aconitine in water acidulated with acetic acid should be injected under the skin of a mouse, over the back portion of one of the hind legs. Under the skin of another mouse a known quantity of aconitine should be injected. The symptoms and the modes of dying displayed by the two mice should be carefully watched and compared. Stevenson finds that $\frac{1}{3000}$ of a grain of crystallised aconitine so injected kills a mouse in eighteen minutes.

III. Kundrat[1] recommends as a test for aconitine a solution of ammonium vanadate (two per cent. strength) in strong

[1] *Chemik. Zeit.*, 1889.

sulphuric acid. This gives with solid aconitine a light coffee-brown colour. This test, however, can only be regarded as of use as a confirmatory test for aconitine, and not as establishing the identity of the alkaloid.

Veratrum and Veratrine

The veratrum plants are *Veratrum Album* and *Veratrum Viride* (Nat. Ord. Melanthaceæ). These plants contain several alkaloids which have been investigated by Wright and the author. Of these the principal are veratrine and cevadine. Commercial veratrine is impure, and is generally a mixture of veratrine and cevadine; it is obtained from sabadilla seeds. Veratrine is a violent sternutatory, a small quantity of it dissipated in the form of dust in the air producing violent sneezing. The action of veratrine is at first that of a stimulant to the motor nerves, quickly followed by paralysis of the motor nerve-endings. The character of muscular contractility is altered in a peculiar manner, the contraction is a prolonged one, and the relaxation is also prolonged. The sensory nerves are similarly affected, being stimulated at first, but afterwards paralysed. The heart is slowed and the blood-pressure lowered from vaso-motor paresis or paralysis. Respiration is quickened at first, then slowed, and finally arrested from paralysis of the respiratory centres, death resulting from asphyxia.

Symptoms.—(i) An acrid burning sensation in the throat with a feeling of constriction; burning pain down the œsophagus to the stomach, vomiting and great thirst; diarrhœa may occur with tenesmus. (ii) Feeble pulse with slowed respiration. (iii) Pupils sometimes dilated, sometimes contracted. (iv) Collapse with pallor, coldness of the surface and twitching of the muscles. (v) Consciousness is generally present till collapse sets in, but occasionally delirium and stupor may supervene early. Convulsions may also occur.

Treatment and antidotes.—The same as for aconite poisoning (see p. 305).

Post-mortem appearances.—Nothing characteristic.

Fatal dose.—Death has taken place after taking about eighteen grains of powdered veratrum root, but recovery has occurred after much larger quantities of the root have been taken. Dixon Mann[1] mentions two cases of recovery under treatment, after four grains and a half and nearly three grains of veratrine respectively had been swallowed.

Analysis and tests.—Veratrine is extracted from the contents of the stomach or from the viscera by the usual process for alkaloidal extraction (see p. 254). To the alkaloidal residue so obtained the following tests may be applied:—

I. A small quantity of the alkaloid in the form of dust produces violent sneezing.

II. A drop of strong sulphuric acid applied to solid veratrine in a porcelain dish produces a yellow tint, changing to reddish, and finally to deep crimson. This colour-change is brought about immediately by heat.

III. If strong hydrochloric acid be added to solid veratrine, and the mixture heated, a red colour is produced.

IV. Sulphomolybdic acid added to solid veratrine produces a reddish colour, changing to dirty brown, greenish, and finally blue.

Black Hellebore

Helleborus Niger (Nat. Ord. Ranunculaceæ) is the *black hellebore* or *Christmas rose* (Plate VII.) It is a drastic purgative, but is little used in this country. As a poison it acts as an irritant to the gastro-intestinal tract.

Symptoms.—(i) Abdominal pain, vomiting and purging. (ii) Cold sweats, collapse, feeble pulse and dyspnœa. (iii) The pupils are generally dilated. (iv) Cramps and convulsions.

Treatment and antidotes.—The stomach should be emptied and washed out by means of the stomach-pump or tube. Warmth should be applied to the surface and stimulants administered. Morphine may be given by hypodermic injections.

[1] *Forensic Med. and Toxicol.*

Plate VII.

Christmas Rose (Helleborus Niger)

Post-mortem appearances.—Nothing characteristic. Inflammation of the mucous membrane of the stomach and intestines is generally found.

Fatal dose.—Nothing definite is known.

Fatal period.—Nothing definite is known.

Analysis and tests.—Helleborin, one of the active principles of black hellebore, can be extracted from an acid solution by agitation with ether. The residue left on evaporation of the ether gives a bright red colour with strong sulphuric acid.

CHAPTER XVIII

Poisoning by cocaine—Cocculus indicus—Conium (hemlock)—Œnanthe crocata (water dropwort)—Cicuta virosa (water hemlock)—Æthusa cynapium (fool's parsley)—Gelsemium (yellow jasmin)—Digitalis (foxglove)—Tobacco.

Cocaine

Cocaine is an alkaloid obtained from the leaves of *Erythroxylon Coca* (Nat. Ord. Erythroxylaceæ). Cocaine exercises a paralysing action on the endings of the sensory nerves, and on that account is used to produce local anæsthesia. When absorbed into the circulation it paralyses the vagus, causing extreme rapidity of the heart's beats. Applied to the eye, or after absorption into the circulation, it produces dilatation of the pupils. It first stimulates, then depresses, and finally paralyses the centres of the brain and spinal cord. It depresses the action of the heart and lowers blood-pressure, and finally produces death from paralysis of respiration, or, according to Mosso, by tetanus of the respiratory muscles. It frequently causes a considerable rise of body temperature. Maurel,[1] who has made a series of experimental researches on the action of poisons on leucocytes, believes that death by cocaine is the consequence of the death of the leucocytes, or of modifications which the latter sustain under the influence of that alkaloid. The results of more recent experiments made by him [2] show that the leucocytes become spherical, rigid, and increased in size under the influence of cocaine; that the capillaries contract, and that thromboses and embolisms, particularly pulmonary embolisms, capable of causing fatal accidents, may be produced.

[1] *Bulletin Thérapeutique*, 1892. [2] *Sem. Méd.*, 1894.

Symptoms.—(i) Extreme pallor and cyanosis, accompanied by cold sweats. (ii) Faintness, and occasionally vomiting. (iii) Pain in the præcordial region. (iv) The pulse is rapid at first, afterwards slowed. (v) Intermittent action of the heart and laboured respiration. (vi) Dilatation of the pupils. (vii) Unsteady gait, confusion of ideas, incoherence of speech, with volubility and temporary delirium, may occur. (viii) Convulsions and unconsciousness may also occur.

Treatment and antidotes.—If convulsions occur the inhalation of chloroform should be resorted to. Brandy should be administered, solution of ammonia should be applied to the nostrils, and ten minims of it given internally every five minutes till the collapse is overcome and the heart is recovering its normal action. The inhalation of three drops of nitrite of amyl has been recommended in cases of cocaine poisoning. It is, however, not of much use, and sometimes does more harm than good.

Post-mortem appearances.—Nothing characteristic. Congestion of the viscera and of the membranes of the brain and spinal cord is generally present, due to vaso-motor paralysis.

Fatal dose.—About two-thirds of a grain injected subcutaneously has caused the death of a woman, aged seventy-one, in five hours (Dixon Mann). Three grains and a half injected into the breast of a woman, preparatory to an operation, produced death in twenty-four minutes. Half a grain of cocaine, injected into the gum of an adult, has produced alarming symptoms, which were relieved by application of ammonia to the nostrils. Gooding[1] relates the case of a negress who developed alarming symptoms after less than half a grain had been injected into the gum; she recovered after hypodermic injections of ether and ammonia.

Cocaine, after absorption into the system, is rapidly eliminated by the kidneys; should there, however, be extensive disease of the kidneys, elimination is more or less prevented,

[1] *The Lancet*, 1888.

and the poison, therefore, remains in the system. In a case[1] in which twenty grains of cocaine in solution were accidentally administered, by the mouth, to a man, death took place about one hour after the administration. At the post-mortem examination it was found that the left kidney was entirely destroyed from disease of long standing, and the other was in a tuberculous condition. Mowat relates the case of a man, in whom serious symptoms of cocaine poisoning were produced by the injection of one-seventh of a grain of cocaine at the sides of a rodent ulcer of the eyelid, previous to operation. The symptoms consisted of pallor, dyspnœa, dilatation of pupils, weak and very feeble pulse, and clonic spasms. Serious effects have also followed the introduction of very minute doses of cocaine into the eye.

Fatal period.—Death has occurred in twenty minutes after hypodermic injection of three grains and a half of cocaine. Persons addicted to morphinism can take large quantities of cocaine, and in the case of a morphine *habitué* twenty-three grains of cocaine have been taken by hypodermic injection in one day. Dixon Mann[2] mentions a case in which recovery took place after forty-six grains of cocaine were taken into the stomach.

Chronic Poisoning by Cocaine, and the Cocaine Habit

The hypodermic injection of cocaine is now, unfortunately, a common habit, the craving for the drug by those who accustom themselves to its use being similar to the craving for morphine. When used in this way, it produces at first agreeable sensations, together with feelings somewhat similar to those of slight intoxication; these are succeeded by palpitation and irregular action of the heart. The constant use of cocaine induces depression and inaptitude for the work and cares of everyday life. Déjerine mentions the case of a young man, aged twenty-six, who, beginning with subcutaneous

[1] *The Lancet*, 1889. [2] *Forensic Med. and Toxicol.*

injections of one-seventh of a grain, gradually increased the quantity until he used seven grains for an injection. The injections produced agreeable sensations and sexual desire, followed by emission. On one occasion he injected fifteen grains of cocaine, and shortly afterwards fell down unconscious. Under treatment the patient recovered.

Analysis and tests.—Cocaine may be separated from the contents of the stomach or from the viscera by the usual process for alkaloidal extraction (see p. 254). To the alkaloidal residue left after the final evaporation of the chloroform and ether mixture, the following tests may be applied :—

I. If a few drops of strong nitric acid are added to a small quantity of solid cocaine, or to one of its salts, and the mixture evaporated to dryness on a water-bath, the residue gives, on stirring with a drop or two of a strong solution of caustic potash in absolute alcohol, a distinct and peculiar odour, recalling that of peppermint, or citronella, or meadow-sweet. Da Silva, who first described this test, considers it to be an extremely delicate one for cocaine, but the odour is scarcely distinctive enough to render the test by itself an absolutely reliable one.

II. If to a few drops of strong sulphuric acid in a porcelain dish some powdered or crystallised resorcin be added, and the mixture moved to and fro a little, the subsequent addition of a small quantity of cocaine produces a splendid blue colouration, changing to a light rose colour on addition of caustic soda. Goeldner [1] states that no other alkaloids give anything approaching to this reaction.

III. If a few drops of a five per cent. solution of chromic acid in water are added gradually to a solution of cocaine hydrochloride, each drop produces a precipitate which immediately redissolves. If now a small quantity of strong hydrochloric acid be added, a heavy yellow precipitate of cocaine chromate is produced. This reaction is stated by Metzer [2] to be peculiar to cocaine.

[1] *Pharm. Zeit.*, 34, 471. [2] *Ibid.*, 697.

IV. The physiological test may be used by applying a solution of cocaine hydrochloride to the tongue or lips, when a feeling of numbness is produced, due to the local anæsthetic action of the alkaloid.

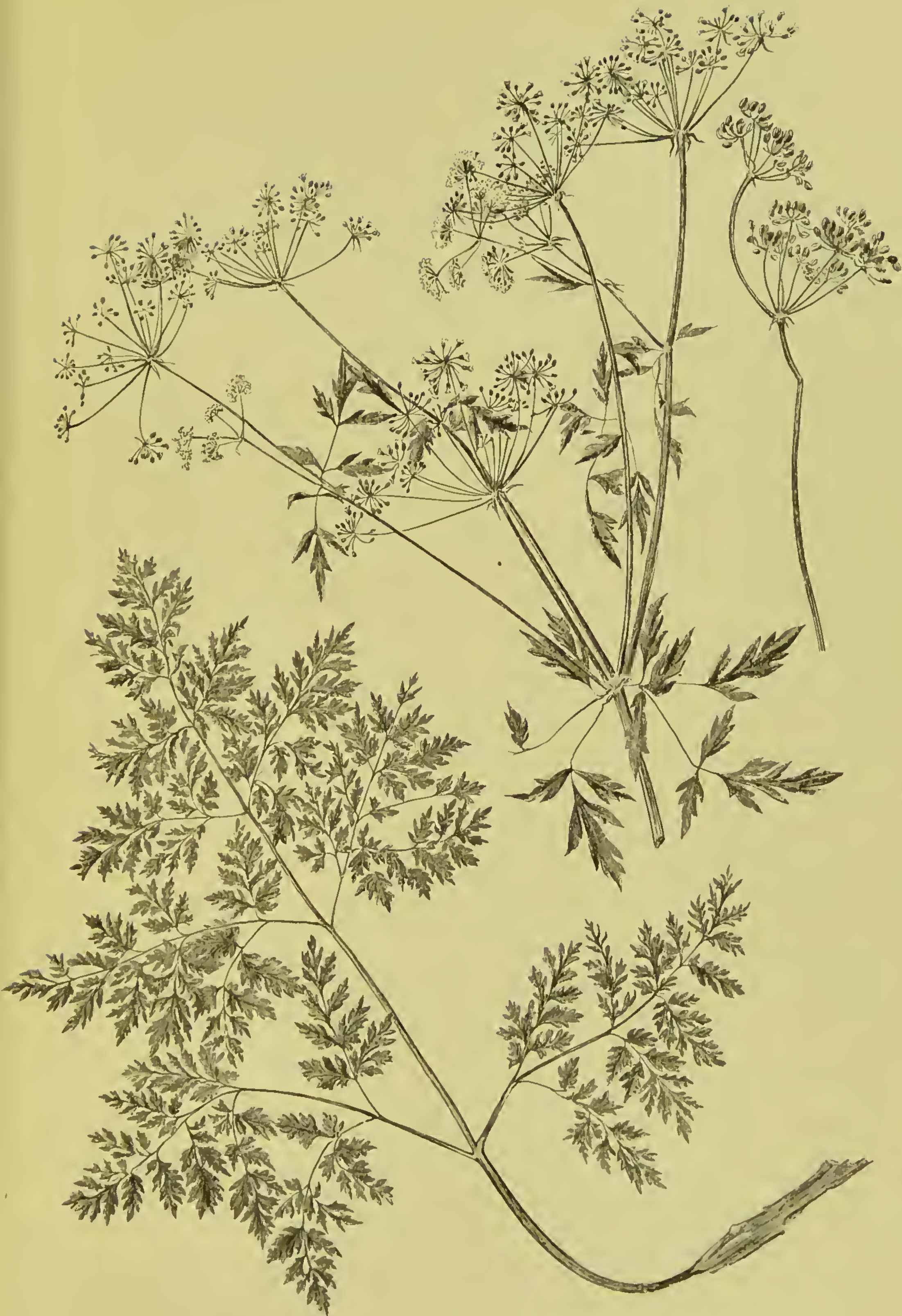
Cocculus Indicus

Cocculus Indicus, or Levant Nut, is the fruit of *Anamirta Cocculus* (Nat. Ord. Menispermaceæ). The kernel of the berry is the poisonous part, and the poisonous properties are due to a neutral crystalline body named picrotoxin, which has an intensely bitter taste. Cocculus indicus is employed as a fish poison for the production of stupefaction, and for the malicious destruction of game. It is also illegally used to adulterate inferior malt liquors, in order to impart a bitter flavour, so that a smaller quantity of hops should be required in the manufacture of the beer, and to give it an intoxicating property, which, from its poorness in alcohol, it would not possess of itself. Cocculus indicus is a gastro-intestinal irritant, and also a stimulant to the cerebro-spinal centres, and on this latter account it possesses a powerfully convulsant action.

Symptoms.—(i) Abdominal pain may occur, accompanied by nausea and vomiting. (ii) Faintness, giddiness, and thirst. (iii) Weak pulse, slow and laboured respiration; unconsciousness, if sufficient of the drug be taken. Convulsions may occur, with opisthotonos, and relaxation between the attacks. The convulsive attacks may be produced reflexly, and bear considerable resemblance to those caused by strychnine. The external application of cocculus indicus or of its preparations may produce poisonous effects. Many of the symptoms mentioned above, together with a fatal result, have ensued from the application of a tincture of cocculus indicus to the scalp of a child for the destruction of vermin.

Treatment and antidotes.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube, or, if these are not procurable, an emetic of mustard and water should be given. To counteract the convulsive seizures,

Plate VIII



Hemlock (Conium Maculatum)

chloral should be used, as in strychnine poisoning (see p. 279), or inhalations of chloroform may be given for the same purpose. Kossa states that paraldehyde is an effectual specific against picrotoxin.

Post-mortem appearances.—Nothing characteristic. The usual signs of gastro-intestinal irritation may be present.

Fatal dose.—Nothing definite is known.

Fatal period.—Death has occurred in half an hour, but may be delayed for several hours.

Analysis and tests.—Picrotoxin can be extracted by means of ether from hydrochloric acid solutions. To the picrotoxin left after evaporation of the ether the following tests may be applied :—

I. If picrotoxin in the solid state be moistened with strong sulphuric acid, it dissolves, giving a beautiful saffron-yellow colouration. On the addition of a small particle of potassium bichromate, the colour changes to violet, passing into brown.

II. If solid picrotoxin be moistened with a little strong hydrochloric acid, the mixture dried on the water-bath, and the residue treated with a drop or two of strong sulphuric acid, on the subsequent addition of an excess of solution of caustic soda a brick-red colour is produced.

III. Picrotoxin may be detected in beer by agitation with freshly precipitated lead hydrate, which forms a stable insoluble compound between the picrotoxin and the lead. If the lead precipitate be collected, washed, and dried, it gives, on moistening with strong sulphuric acid, the saffron-yellow colour referred to above. The lead hydrate must be freshly prepared and well washed previous to use.

Conium or Hemlock

This poisonous plant is the *Conium Maculatum* (Plate VIII.), or *spotted hemlock* (Nat. Ord. Umbelliferæ). All parts of the plant are poisonous. Conium is the poison by which Socrates perished. Poisoning by conium is generally the result of

accident, the fresh leaves being occasionally used in soups in mistake for parsley. Conium very closely resembles in appearance 'Fool's Parsley' (*Æthusa Cynapium*) (Plate IX.). The entire plants may be distinguished from one another by the dark purple spots which are found on the thicker portions of the stem of the conium or spotted hemlock, whereas no such spots are present on the stem of fool's parsley. These spots are not found on the stems or branches of conium if they are much smaller than the little finger; but, in the absence of the thicker portions of the stem, the two plants can be distinguished by the characters of the involucels around the flowers. In the case of conium, the involucels consist of very short bracts arranged around the flowers, which, at first sight, are scarcely visible; whereas, in the case of fool's parsley, the involucels consist of long, slender, pendent bracts, which hang down on one side of each little group of flowers (see Plates VIII. and IX.) The active principles of conium are conine and methyl-conine. Conine is a liquid volatile alkaloid, and in the pure state is a clear, oily fluid, possessing a peculiar, unpleasant odour. When diluted with water, conine has a very characteristic smell, resembling very closely that of mice; but in the pure condition the odour is more like that of the liquid found in a foul tobacco pipe. The motor nerves are especially attacked by conium, being paralysed from their peripheral terminations upwards to the nerve-centres. Death is due to asphyxia from paralysis of the respiratory nerves and depression of the respiratory centre. Methyl-conine abolishes the reflex of the spinal cord.

Symptoms.—(i) Nausea, abdominal pain, vomiting, and diarrhœa. (ii) Headache, imperfect vision, dilated pupils. (iii) Sensation of weight in the extremities, with increasing muscular weakness, followed by paralysis spreading from the legs to the trunk, the arms not being so rapidly affected as the other parts. (iv) Dyspnœa, with gradual slowing of the respiratory movements. (v) Dysphagia and drowsiness. (vi) Convul-

sions, coma, and even delirium, if death be delayed. The convulsions are generally asphyxial in their nature.

Treatment and antidotes.—The stomach should be emptied by means of the stomach-pump or stomach-tube. If these are not available, an emetic of mustard and water should be given. Stimulants and external warmth should be employed, and strychnine injected hypodermically. Artificial respiration should be resorted to, if necessary; in severe cases, if persisted in, it may be the means of saving the life of the patient.

Post-mortem appearances.—Special search should be made in the stomach and intestines for the leaves and fruits of the conium plant, which should then be identified by careful examination with a lens or low power of the microscope. There are no characteristic post-mortem appearances. Redness of the mucous membrane of the stomach, and congestion of the lungs, are generally present.

Fatal dose.—One drop of conine is considered to be a poisonous dose, but no case has actually occurred to demonstrate this.

Fatal period.—Death usually occurs in from one to three hours.

Analysis and tests.—Conine is extracted from the contents of the stomach or intestines, or from the viscera by the usual process for alkaloidal extraction (see p. 254). On evaporation of the final chloroform and ether extract at a low temperature, the liquid alkaloid conine is left. To it the following tests may be applied.

I. *The odour* (see above).—Too much reliance should not be placed upon the odour of conine. Harley observes that caustic potash in contact with organic substances may frequently develop an odour which might be mistaken for that of conine, when the latter is absent.

II. If conine be warmed with potassium bichromate and dilute sulphuric acid, butyric acid is produced, which may be recognised by its characteristic odour.

III. If conine be dropped into a solution of alloxan, the

latter is coloured an intense reddish-purple after a few minutes. On standing, white needle-shaped crystals separate out. These, if collected and dissolved in cold caustic potash solution, produce a bluish-purple colour, and emit a strong odour of the conine.

Œnanthe Crocata

Œnanthe Crocata, or *water dropwort* (Nat. Ord. Umbelliferæ), is one of the most poisonous of the umbelliferous plants. The roots have been eaten in mistake for parsnip, and the plant has also been mistaken for celery. The plant is a very fatal one to animals, and oxen have not unfrequently been killed by eating it. Cameron[1] reports that forty-three oxen, after feeding upon some of the œnanthe crocata, were seized with foaming at the mouth, shivering, difficult breathing, spasms, and finally death occurred in all the cases.

Symptoms.—(i) Abdominal pain, vomiting, and diarrhœa. (ii) Slow feeble pulse, dilated pupils, collapse, and cyanosis. (iii) Delirium, insensibility, and convulsions.

Post-mortem appearances.—The contents of the stomach and intestines should be carefully examined for portions of the plant. There are no characteristic post-mortem appearances; as a rule, signs of inflammation of the stomach and intestines are present.

Treatment.—The same as in cases of conium poisoning (see p. 319).

Cicuta Virosa

Cicuta Virosa, or *water hemlock* (Nat. Ord. Umbelliferæ), is also one of the poisonous umbelliferous plants. The roots have been eaten in mistake for parsnip.

Symptoms.—These are somewhat similar to those of conium poisoning, and consist of vomiting, muscular twitching, a feeling of numbness, paralysis, laboured breathing, convulsions, and unconsciousness.

Treatment.—The same as for conium poisoning (see p. 319).

[1] *The Lancet*, 1873.

Plate IX.

Fool's Parsley (Æthusa Cynapium)

Æthusa Cynapium, or Fool's Parsley

Æthusa Cynapium (Plate IX.), or *fool's parsley*, or *lesser hemlock* (Nat. Ord. Umbelliferæ), is also one of the umbelliferous plants, the leaves of which have been gathered by mistake for parsley. The symptoms ascribed to poisoning by this plant are somewhat similar to those of poisoning by conium, but the experiments of Harley throw considerable doubt upon the possession of any poisonous properties by fool's parsley. After performing a number of experiments upon himself and on patients with the juice and various preparations of the plant, he came to the conclusion that it did not possess any poisonous nature. Quantities equivalent to three ounces of the fresh juice, and to six ounces of fresh herb, were administered at one time without the production of any poisonous effects. Probably, in cases of alleged poisoning by Æthusa Cynapium, other poisonous umbelliferous plants may have been mistaken for it.

Gelsemium

Gelsemium Sempervirens is the *yellow jasmine* or *jessamine* of North America. A tincture of the root is largely used in the treatment of neuralgia. Fatal results have occasionally resulted from the use of gelsemium. The symptoms somewhat resemble those produced by veratrine. Gelsemine, the active alkaloid, is a powerful mydriatic if dropped into the eye. It also exercises a paralysing effect on the spinal cord and respiratory centres.

Symptoms.—(i) Dimness of vision, dilated pupils, and paralysis of the eyelids. (ii) Impairment of speech, and general prostration. (iii) Small and frequent pulse, laboured breathing. (iv) Trismus, clonic spasms, and unconsciousness may occur.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube. If neither of these be procurable, an emetic of mustard and water should be administered. Stimulants such as brandy or ether should be employed. Hypodermic injections of morphine have

been recommended; on the other hand, the use of atropine or strychnine hypodermically as a respiratory stimulant has been advised. Foy[1] found that nitroglycerine was a rapid and perfect antidote.

Post-mortem appearances.—Nothing characteristic.

Fatal dose.—Half an ounce of the tincture of gelsemium and three teaspoonfuls of the fluid extract have respectively caused the death of an adult; thirty-five drops of the tincture have caused the death of a young child.

Fatal period.—One hour is the shortest known fatal period; several hours may elapse before death occurs.

Analysis and tests.—Gelsemine may be extracted by the usual process for alkaloidal extraction (see p. 254). Gelsemine behaves almost exactly like strychnine in its colour reactions with sulphuric acid and an oxidising agent, such as manganese dioxide or potassium bichromate. It may be distinguished from strychnine by the two following tests:

I. Gelsemine gives, with strong sulphuric acid alone, a green colour changing to red; strychnine is unaffected by sulphuric acid alone.

II. Gelsemine gives, when evaporated with strong nitric acid, a bluish-green colour; strychnine does not.

Digitalis

Digitalis Purpurea (Plate X.), or *foxglove* (Nat. Ord. Scrophulariaceæ), is a plant growing wild in this country and in most of the countries of Northern Europe. Its chief poisonous properties are due to a glucoside digitalin, and in addition it contains digitoxin, digitonin, and digitalein. Of these, digitoxin, according to Kopp, is from six to ten times stronger in its toxic effect than digitalin. In poisonous doses digitalis acts as a heart poison, causing death by paralysis of the heart.

Symptoms.—(i) Abdominal pain, nausea, vomiting, and occasionally purging. (ii) Surface cold and clammy, vertigo,

[1] *Med. Press and Circ.*, 1886.

Plate X.

Foxglove or Digitalis (Digitalis Purpurea.)

disordered vision, dilated pupils, prominent eyes, and noises in the ears. (iii) The pulse is generally full and slow if the patient remain in the horizontal position, but becomes rapid and feeble if the patient sit up. The mere raising of the head may produce a feeling of syncope, and the standing upright of the patient may bring on an immediately fatal attack of syncope. (iv) Delirium, stupor, or convulsions are apt to come on just before the occurrence of death. It should be borne in mind that the liability to syncope remains for some days after the initial effects of the poison have passed away.

Treatment and antidote.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube. If neither of these be procurable, an emetic of mustard and water should be administered. External warmth should be applied and stimulants given. Possibly aconitine might be of service as an antidote in cases of digitalis poisoning; certainly digitalis is a useful antidote in cases of aconite poisoning; and, when the heart of a frog has almost ceased to beat under the influence of digitalis, its movements may be restored by aconitine. Up to the present, however, no case of digitalis poisoning has been recorded in which aconitine has been tried as an antidote. After the immediate effects of the digitalis have passed away, the patient should be kept in the recumbent position for several days, on account of the liability to syncope.

Post-mortem appearances.—Nothing characteristic. Inflammation of the mucous membrane of the stomach and intestines may be present. Careful search should, however, be made for portions of the leaves in the contents of the stomach and intestines.

Fatal dose.—Thirty-eight grains of the powdered leaves and nine drachms of the tincture of digitalis have proved fatal. Probably from a quarter to half a grain of digitalin would prove fatal to an adult.

Fatal period.—Death has occurred in three-quarters of an hour after swallowing a large dose of digitalis. As a rule,

twenty-four hours, or more, may elapse before the occurrence of a fatal event.

Analysis and tests.—Digitalin can be extracted from acid solutions by means of chloroform; impurities should be previously removed by agitation with petroleum ether, which does not extract the digitalin. To the digitalin, left after the evaporation of the chloroform, the following tests may be applied.

I. *Physiological test.*—This consists in observing and comparing the action of digitalin, and of the poison suspected to be digitalin, upon the hearts of frogs, noting the gradual irregularity and slowing of the heart beats, together with the manner of the final cessation of the beating of the two hearts. This test was resorted to by Tardieu in 1864 for the detection of digitalin in the *Pommerais case*, in which Dr. de la Pommerais was tried for the murder of a woman with digitalin. Tardieu exposed the hearts of three frogs. The first was left so that the heart was constantly moist, and no poison was administered. Into the pleural sac of the second, a few drops of a dilute solution of digitalin were injected. Into the pleural sac of the third a solution of the suspected poison obtained from the body of the deceased was injected. Tardieu found that the heart of the frog poisoned with the unknown extract behaved in a very similar and comparable manner to that of the frog poisoned with digitalin; whereas the heart of the unpoisoned frog showed at the end of thirty-one minutes very little change, although the hearts of the other two frogs had ceased to beat after the lapse of that time. Fagge and Stevenson,[1] from experiments made with digitalin upon the frog's heart, have shown that the ventricle ultimately stops in the white contracted state. Lauder Brunton also considers the action of digitalin on the heart to consist in the prolongation of the systole.

II. Strong sulphuric acid in the cold imparts to digitalin a brownish colour gradually changing to red; on warming, the colour passes to brown. If this brown liquid be allowed to

[1] *Guy's Hospital Repts.*, 1866.

cool and excess of water added, the colour changes to green, and a green powder is deposited, the liquid gradually assuming a yellowish colour (Tardieu).

III. If digitalin be dissolved in a few drops of strong sulphuric acid, on the addition of bromine water a violet-red colour is produced.

IV. Strong nitric acid imparts to digitalin an orange-red colour, an effervescence of red fumes occurring at the same time.

Tobacco and Nicotine

Tobacco consists of the dried leaves of *Nicotiana Tabacum* (Nat. Ord. Solanaceæ). Tobacco owes its active properties to nicotine, which it contains in combination with malic, citric, and acetic acids. The amount of nicotine present in tobacco varies from two to eight per cent. Nicotine is a liquid volatile alkaloid. In the pure condition it is a colourless oily liquid, and an extremely active poison. In poisonous doses nicotine first stimulates and finally paralyses the nervous system.

Symptoms.—(i) A burning sensation in the throat, followed by nausea, pain in the stomach and vomiting; severe purging sometimes occurs. (ii) Great prostration and giddiness, frequent and feeble pulse, cold clammy skin, trembling, and loss of power of the limbs. (iii) Laboured respiration. The pupils may be either unaffected, contracted, or dilated. (iv) Loss of consciousness, and tetanic convulsions occasionally occur.

Accidents have happened to children from playing with old tobacco pipes. A child,[1] aged three, used an old tobacco pipe with which to blow soap bubbles; symptoms of poisoning followed, and the child died in three days. A fatal result has followed the administration of tobacco juice, as obtained from pipes, in the form of a drink. A decoction of tobacco applied to the skin of a man, for an eruptive disease, has caused death in three hours. Tobacco administered in the form of an enema has also caused death.

[1] *Pharm. Jour.*, 1877.

Treatment and antidote.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube; if neither of these be procurable, an emetic of mustard and water should be administered. Stimulants should be given, external wamth applied, and artificial respiration resorted to if necessary. Hypodermic injections of strychnine should be used to counteract the paralysing effect of the poison.

Post-mortem appearances.—The odour of the poison may be detected in the contents of the stomach, or in the vomited matters. Inflammation of the mucous membrane of the stomach and intestines is generally present, together with, as a rule, congestion of the liver, lungs, and brain. The blood is usually dark and liquid.

Fatal dose.—Probably from one to three drops of pure nicotine would be fatal to an adult in a few minutes. In the combustion of tobacco for smoking purposes, most of the nicotine is converted by the heat into pyridine bases. It was formerly doubted whether any nicotine was present in tobacco smoke, but the researches of Melsens and Kissling have proved its presence in a proportion equal to about one-seventh of that present in the original tobacco, the remaining six-sevenths being converted principally into pyridine bases. Fatal poisoning has resulted from tobacco smoking. A boy smoked a penny-worth of twist tobacco and afterwards became very sick; some hours later he was found dead in bed.[1] Blyth mentions the occurrence of death in the case of two brothers who smoked from seventeen to eighteen German pipefuls of tobacco.

Fatal period.—Dixon Mann mentions a fatal case of nicotine poisoning in which death occurred in three or four minutes. In the case of Count Bocarmé, who poisoned his wife's brother, Fougnies, with nicotine, death took place in five minutes. An enema of tobacco has caused death in fifteen minutes. Snuff swallowed in whisky has caused death in one hour.

Analysis and tests.—Nicotine can be extracted by the

[1] *The Lancet*, 1885.

usual process for alkaloidal extraction (see p. 254), and is left as an oily liquid on evaporation of the final chloroform and ether extract at low temperatures. To the liquid so obtained the following tests may be applied, after dissolving it in dilute hydrochloric acid.

I. Solution of mercuric chloride gives a white crystalline precipitate changing to yellow. The crystals when viewed under the microscope show a beautiful and characteristic stellate appearance, the different crystals exhibiting various patterns.

II. If a solution of iodine in ether be added to an ethereal solution of nicotine, long needle-like crystals form after some hours.

III. On the addition of a solution of caustic potash to a solution of nicotine hydrochloride, a strong tobacco-like odour is developed on warming the solution.

Nicotine is apt to be mistaken for conine, but can be distinguished by (*a*) mercuric chloride solution, yielding a crystalline precipitate with nicotine, and an amorphous precipitate with conine; (*b*) platinic chloride, which throws down a yellow precipitate, becoming crystalline on standing in the case of nicotine, but yielding no precipitate with conine.

CHAPTER XIX

Poisoning by lobelia—Colchicum—Stavesacre—Laburnum—Mezereon—Yew—Primula—Horse-chestnut—Tansy—Jaborandi—Calabar bean—Nutmeg—Curare—Savin—Male fern—Santonin.

Lobelia

Lobelia Inflata is also known as *Indian tobacco* (Nat. Ord. Lobeliaceæ). The leaves and seeds are the parts used. They owe their activity to an alkaloid lobeline, which is a yellowish liquid. In poisonous doses lobelia acts as a gastro-intestinal irritant, and as a depressant. It is a drug that has been, and is still, used by ignorant quacks and herbalists.

Symptoms.—(i) Nausea and vomiting; purging sometimes occurs. (ii) Cold sweats, prostration, small feeble pulse, contracted pupils. (iii) Stupor, occasionally convulsions, coma.

Treatment and antidote.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube, or, if neither of these be procurable, an emetic of mustard and water should be administered. Stimulants should be given, and warmth applied externally. Hypodermic injections of strychnine may be employed to counteract the depressing effects of the drug.

Post-mortem appearances.—These are very similar to those caused by tobacco (see p. 326).

Fatal dose.—One drachm of the powdered leaves has proved fatal to a male adult.

Fatal period.—Death has occurred half an hour after taking an infusion of lobelia.

Analysis and tests.—Lobeline may be extracted in a similar

Plate XI.

Meadow Saffron or Colchicum (Colchicum Autumnale)

Danielsson & Co. sculp.

way to nicotine. To the liquid alkaloid so obtained the following tests may be applied :—

I. Strong sulphuric acid produces with lobeline a red colour.

II. Lobeline gives with sulphomolybdic acid a violet colour. A similar colour is given by morphine, but morphine is a solid substance, lobeline a liquid; moreover, morphine does not give a red colouration with strong sulphuric acid, as lobeline does.

Colchicum

Colchicum Autumnale (Plate XI.) is the *meadow saffron* (Nat. Ord. Melanthaceæ). The corm, or underground stem, and the seeds are the parts used in medicine. The active principle of colchicum is an alkaloid colchicine. In large doses colchicum is a powerful gastro-intestinal irritant.

Symptoms.—(i) Burning pain in the throat and stomach. (ii) Great thirst, vomiting, and purging, with severe colicky pains. (iii) Collapse, cramps, rapid and feeble pulse, dilated pupils, pale or cyanosed face. (iv) Muscular spasms and convulsions sometimes occur. The mind is usually clear to the last, but delirium followed by stupor may occasionally supervene.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube; if neither of these be procurable, an emetic of mustard and water should be given. Stimulants should be administered, warmth applied externally, and artificial respiration carried on, if necessary. If the colicky pains are severe, a hypodermic injection of morphine should be used to relieve them.

Post-mortem appearances.—Nothing characteristic; inflammation of the mucous membrane of the stomach and intestines is generally present, and congestion of the lungs has also been noticed.

Fatal dose.—One tablespoonful of the seeds, forty-eight grains of the dried corm or bulb, and three teaspoonfuls and a

half of colchicum wine have respectively proved fatal. Reese[1] states that less than half a grain of colchicine has proved fatal. On one occasion a woman, aged forty-three, swallowed about six grains of colchicine, and died in thirty-one hours.

Fatal period.—Death has occurred in seven hours, and has been delayed for several days; it generally occurs within twenty-four hours.

Analysis and tests.—Colchicine is extracted from an acid solution by agitation with chloroform. To the residue left on evaporation of the chloroform, the following tests may be applied:—

I. Strong nitric acid gives with colchicine a violet colour, changing to blue and brownish-green. If the violet solution be diluted with water, it changes to a yellow colour, which, on the addition of solution of caustic soda, becomes orange-yellow or red.

II. Strong sulphuric acid gives with colchicine a yellow colour, which, on the addition of a drop of nitric acid, changes to green, violet, and finally reddish-brown.

Colchicine resembles veratrine in many of its properties, but the two alkaloids may be distinguished by the different colour reactions given with strong sulphuric acid.

Stavesacre

The seeds of *Delphinium Staphisagria*, or *stavesacre* (Nat. Ord. Ranunculaceæ), contain two poisonous principles, delphinine and staphisagrine. The seeds may act as a gastro-intestinal irritant.

Symptoms.—Feeble pulse, collapse, with cold surface, laboured breathing, and dilated pupils.

Treatment.—The stomach should be emptied and washed out, or emetics should be employed.

Laburnum

Cytisus Laburnum (Nat. Ord. Leguminosæ) is the common *laburnum tree*, grown so extensively in gardens and parks in

[1] *Med. Jurispr. and Toxicol.*, 1891.

this country. All parts of the plant are poisonous, and cases of poisoning have arisen from persons chewing and swallowing portions of the flowers, seeds, and roots. The poisonous properties of laburnum are due to an alkaloid cytisine. Cytisine is an irritant narcotic, and exercises a stimulating action at first upon the nervous system, followed by a paralysing effect. If death occur, it is due to asphyxia, from paralysis of the respiratory centres. Death has resulted from swallowing both the bark and the seeds. Poisoning by laburnum especially occurs among children from eating or chewing the root or seeds, which possess a sweetish taste.

Symptoms.—(i) A hot feeling in the throat, followed by thirst and epigastric pain. (ii) Vomiting and diarrhœa. (iii) Generally collapse occurs, with a cold moist surface, a feeble irregular pulse, and laboured respiration. (iv) The pupils are usually dilated, but are occasionally contracted. (v) Prostration, drowsiness, and coma supervene; delirium and convulsions also occasionally occur.

St. Johnston [1] describes six cases of laburnum poisoning occurring in children whose ages ranged from five to eleven years. The symptoms of poisoning in some of them were very severe, and were produced by eating from one to ten seeds by individual children. Taylor mentions two cases of poisoning in children from eating laburnum flowers; in both cases recovery took place.

Treatment and antidote.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube, or, if neither of these be available, an emetic of mustard and water should be given; stimulants should be administered, and external warmth applied. To counteract the paralysing effect of the poison, hypodermic injections of strychnine should be given. Artificial respiration must be resorted to if necessary.

Post-mortem appearances.—Nothing characteristic.

Fatal dose.—Nothing definite is known. Stewart [2] mentions the case of a child, aged two years and a half, who

[1] *Brit. Med. Jour.*, 1891. [2] *Ibid.*, 1888.

swallowed from sixty to seventy laburnum seeds, which were rejected after the administration of emetics; in the course of a few hours the child completely recovered.

Fatal period.—Death has occurred within an hour, and, on the other hand, has been delayed for seven days.

Analysis and tests.—The alkaloid cytisine can be isolated by the general process for alkaloidal extraction (see p. 254). Chloroform is the best solvent of cytisine, and should be employed in place of the chloroform and ether mixture. To the residue left on the final evaporation of the chloroform extract, the following tests may be applied:—

I. If some of the cytisine be dissolved in a few drops of very dilute hydrochloric acid, and ferric chloride added, a red colour is produced. On the addition of a few drops of peroxide of hydrogen solution, the red colour disappears, and, on warming the colourless liquid, a blue colour is produced.

II. If cytisine be dissolved in a few drops of strong sulphuric acid in the cold, and a drop of nitric acid added, a yellow colour is produced.

III. If cytisine be dissolved in a few drops of strong sulphuric acid in the cold, and a fragment of potassium bichromate added, a yellow colour is produced, changing to brown, and finally to green.

Mezereon

Daphne Mezereum (Nat. Ord. Thymelaceæ) is commonly grown in gardens. The berries are occasionally the cause of accidental poisoning among children; the juice contains a powerful irritant and acrid substance.

Symptoms.—The symptoms produced by eating mezereon berries are as follows: (i) Burning pain in the mouth and throat. The mucous membrane of the mouth, tongue, and throat may, after a few hours, become whitened in appearance, or be raw and swollen. Vomiting generally occurs. (ii) Prostration,

pallor of the face, dilated pupils, and feeble pulse. (iii) Drowsiness and convulsions may supervene.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube. If neither of these be available, an emetic of mustard and water should be given. This treatment should be followed by a dose of castor oil to expel any of the berries from the intestines. Stimulants and external warmth should be employed.

Fatal dose.—Linné records an instance of a little girl who died after eating twelve berries.

Yew

The *yew*, or *Taxus Baccata* (Nat. Ord. Taxaceæ), is one of the coniferous plants. The leaves and berries are the principal poisonous parts of the plant, and owe their properties to the presence of an alkaloid taxine. This substance is an irritant narcotic poison, and death is due to paralysis of the respiratory centres. A decoction of yew is occasionally employed by ignorant people with the object of bringing on menstruation, or of procuring abortion. It is not an uterine stimulant, and frequently, when taken for either of these purposes, has caused death.

Carruthers states that children can eat the fleshy parts of the berry with impunity, but that when the thin crust surrounding the seed is broken, and the seed itself crushed and swallowed, fatal results have ensued. Several deaths have occurred amongst animals from eating the foliage of yew trees. According to Stuart Wortley, taxine, the poisonous principle, is contained chiefly or entirely in the male yew. The experiments of Munro, however, do not entirely confirm this, as, although he obtained a greater quantity of crude taxine from the male leaves than from the female, he is of opinion that the successive stages of purification may have caused an actual alteration in composition.

Symptoms.—(i) Abdominal pain, vomiting, and collapse,

accompanied by dilated pupils and pallor of the face. (ii) Vertigo, laboured breathing, small and irregular pulse. (iii) Spasms, convulsions, and occasionally delirium.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube, or if neither of these be procurable, an emetic of mustard and water should be administered. Stimulants and external warmth should be employed, and an aperient, such as castor oil, should be given to remove any of the parts of the plant that may have passed into the intestines.

Post-mortem appearances.—Careful search should be made for fragments of the leaves and seeds. Apart from the finding of these, there is nothing characteristic of yew poisoning; inflammation of the mucous membrane of the stomach has been found.

Fatal dose.—Taylor mentions a case of a lunatic woman who died after eating some bits of yew leaves. The portions of the leaves taken from the vomited matters, and found in the stomach and intestines after death, did not amount to a teaspoonful, thus demonstrating the fact that yew leaves may in a small quantity prove a fatal poison.

Primula Obconica

Primula Obconica is a variety of primrose, the blossoms of which are used in funeral wreaths. The leaves of this plant applied to the skin are capable of producing a dermatitis, which is very liable to be mistaken for acute eczema, or for erysipelas. In the cases of gardeners working with the plant, the parts first attacked are the back of the hands and fingers, and the eruption may extend thence to the forearms, or the irritant substance may be transferred by means of the fingers to other parts, such as the eyes, mouth, and nostrils. The rash produced may be either a diffuse erythema, or it may occur in red urticarial patches, or it may take the form of small papules on a raised base. The irritant properties seem to reside chiefly in the

leaves, on the surface of which there are glandular hairs, which probably contain some secretion that is the cause of the mischief. With some people, the touch of the leaves is sufficient to produce the eruption. Ackland [1] mentions the case of a lady, who, on passing a bunch of the leaves lightly around the middle of the forearm, was able to produce an eruption consisting of small papules on a raised base; the eruption appeared about twelve hours after contact with the leaves, and produced almost intolerable itching. Amongst gardeners the plant is well known to affect certain individuals. Pooley [2] mentions the case of a gardener who did not suffer any inconvenience from handling the *Primula Obconica* until after an attack of erysipelas, which he considers rendered the patient susceptible to the peculiar influence of the plant.

Horse-Chestnut

The green rind of the horse-chestnut is occasionally eaten by children, and may produce poisonous symptoms. Its active properties are due to a poisonous substance named æsculin. The following symptoms have been produced by eating the green rind of the horse-chestnut: flushed face, full pulse, widely dilated pupils, drowsiness, and delirium. The treatment consists in emptying the stomach by means of the pump or tube, or by an emetic.

Tansy

Tanacetum Vulgare, or tansy, is a herb belonging to the Nat. Ord. Compositæ. The leaves and volatile oil have a reputation among ignorant people as an emmenagogue and ecbolic. Preparations of the plant are also used as an anthelmintic. Tansy is poisonous, and death has resulted from the use of its oil.

Symptoms.—(i) Collapse, cyanosis (the cheeks have occasionally been flushed), dilated pupils, restlessness. (ii) The pulse and respirations are quickened. (iii) Convulsions.

[1] *The Lancet*, 1893. [2] *Ibid.*

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube, or an emetic should be administered; stimulants and external warmth should be employed.

Post-mortem appearances.—The characteristic odour of the oil can be detected in the stomach and intestines, and usually on opening the cavities of the body.

Fatal dose.—Half an ounce of the oil of tansy has killed a pregnant woman within two hours. The dose was taken with the object of bringing on abortion, but death occurred without expulsion of the contents of the uterus. About eleven fluid drachms of the oil have also caused the death of a woman. A drachm and a quarter has caused severe symptoms, from which recovery eventually took place.

Jaborandi and Pilocarpine

Jaborandi consists of the dried leaflets of *Pilocarpus Pennatifolius* (Nat. Ord. Rutaceæ). The active properties are due to an alkaloid pilocarpine. Jaborandi and pilocarpine are powerful sialagogues and diaphoretics, considerably increasing the production of saliva and sweat. In addition, pilocarpine stimulates the motor nerves of the involuntary muscles, and produces, as a rule, contraction of the pupils and increased peristalsis.

Symptoms.—(i) Flushing of the surface followed by copious perspiration, salivation, increased secretion of mucus in the nose and bronchial tubes, lachrymation. (ii) Cardiac oppression, pulse quickened at first, dyspnœa. (iii) Nausea, vomiting, and great thirst. (iv) Contracted pupils, with spasm of accommodation. (v) Prostration, giddiness, occasionally mental disturbance, which is generally accompanied by hallucinations.

Treatment and antidotes.—If the poison has been taken by the mouth, the stomach should be emptied by means of the stomach-pump or stomach-tube, or an emetic of mustard and water should be administered. To counteract the effects of the absorbed poison, atropine should be employed as an antidote,

$\frac{1}{50}$ to $\frac{1}{30}$ of a grain of atropine sulphate being injected hypodermically in successive doses until the pupils begin to dilate, and the excessive sweating diminishes; or the same effect may be produced by administration of tincture of belladonna in twenty-five minim doses by the mouth. If there be much prostration, stimulants should be administered and warmth applied.

Calabar Bean and Physostigmine or Eserine

Calabar Bean is the *ordeal bean* of Calabar, and consists of the seeds of *Physostigma Venenosum* (Nat. Ord. Leguminosæ). Calabar bean owes its active properties to an alkaloid physostigmine, also named eserine, which is used in ophthalmic practice for producing contraction of the pupil. As a poison, calabar bean, or its active principle eserine, acts as a spinal depressant, producing paralysis. Death is due to paralysis of the respiratory centres, and consequent asphyxia. It also causes contraction of the pupil, laboured breathing, and occasionally convulsions.

Symptoms.—(i) Giddiness, faintness, epigastric pain, and usually vomiting. (ii) Prostration, feeble action of the heart, with a small slow pulse, and cold and moist surface. (iii) Pupils generally contracted, with spasm of accommodation. (iv) The mind is generally clear, but drowsiness, and even unconsciousness, occasionally occur. Leibholz [1] records the cases of two girls who each swallowed in solution three-quarters of a grain of physostigmine sulphate; half an hour later they suddenly became unconscious, and the pupils were widely dilated. This dilatation lasted more or less for several days, and recovery eventually took place. These two cases are peculiar, as dilatation of the pupils was recorded for the first time in connection with physostigmine poisoning. Occasionally eserine (physostigmine) has produced symptoms of poisoning after a solution of it has been dropped into the eyes for ophthalmic purposes.

[1] *Vierteljahrsschr. f. ger. Med.*, 1892.

Treatment and antidotes.—If the poison has been taken by the mouth, the stomach should be emptied and washed out by means of the stomach-pump or tube, or an emetic of mustard and water should be administered. To counteract the effects of absorbed poison, atropine should be employed as an antidote, $\frac{1}{50}$ to $\frac{1}{30}$ of a grain of atropine sulphate being injected hypodermically in successive doses until the pupils begin to dilate; or the same effect may be produced by the administration of tincture of belladonna in twenty-five-minim doses by the mouth. If there be much prostration, stimulants should be administered, and warmth applied; artificial respiration should be resorted to if necessary.

Fatal dose.—Six beans have proved fatal to a boy aged six.

Analysis and tests.—Eserine may be extracted by the general process for alkaloidal extraction (see p. 254), benzene being employed as an ultimate solvent in place of the chloroform and ether mixture. To the residue obtained on evaporation of the benzene extract, the following tests may be applied:—

I. If an aqueous solution of a salt of eserine be heated to boiling, and then strong nitric acid added, a yellowish or orange solution is obtained. This colour changes on addition of excess of caustic soda to a beautiful violet. On acidulating the solution the violet colour is discharged, to be again restored on addition of more alkali.

II. If a small quantity of eserine, or one of its salts, be evaporated in the presence of excess of solution of ammonia, a blue residue is obtained. This, dissolved in dilute acids, produces a red-coloured liquid, which, in reflected light, exhibits a beautiful red fluorescence.

III. Bromine water added to a solution of eserine produces a red or orange coloured turbidity, which clears up on warming.

IV. *Physiological test.*—This consists in introducing a drop of an aqueous solution of eserine into the eye of a rabbit or small cat, when contraction of the pupil is produced. This test may be repeated on the human eye.

Nutmeg

Nutmeg, which is commonly used as a flavouring agent, is harmless in small quantities, but if taken in excessive quantities it may produce poisonous effects. In poisonous doses it may produce either drowsiness, which may pass into unconsciousness, or, on the other hand, it may act as a deliriant. A few cases of nutmeg poisoning—none of them fatal—have been recorded. Sawyer [1] describes a case of a boy, three years old, who ate portions of five nutmegs; he became dizzy, and finally unconscious, and slept for thirty consecutive hours. Bentlip [2] records a case of a farmer, who took one evening the whole of a nutmeg grated up and mixed with milk; early the next morning he was seized with giddiness, inability to stand, great pain in the head, great thirst, numbness of the limbs, somewhat contracted pupils, and inability to distinguish objects around him. After the administration of ten grains of calomel, followed two hours later by an ounce of castor oil, the patient rapidly recovered. Reading [3] relates a case of a lady, three months pregnant, who took three powdered nutmegs with the idea of procuring an abortion. The symptoms consisted of vomiting and delirium, accompanied by hallucinations; intermittent delirium continued for twenty-four hours, when she recovered without abortion having taken place. Simpson [4] reports a case of a woman, aged twenty-six, who took two nutmegs, bruised, in a small quantity of gin, with the object of procuring a miscarriage. Similar symptoms to those above described occurred The woman recovered, free vomiting having taken place.

Curare

This substance is an extract prepared from the bark of *Strychnos Toxifera*, a native of Guiana. The extract, which is a black shining brittle mass, is the Indian arrow poison, and

[1] *New York Med. Jour.*, 1889.
[2] *Brit. Med. Jour.*, 1889.
[3] *Therap. Gaz.*, 1892.
[4] *The Lancet*, 1895.

is prepared by different tribes of Indians in South America. Curare is also known under the names of *curari*, *wourali*, *woorara*, and *urari*. Curare exerts both a paralysing and tetanising action, but its principal poisonous properties are due to its paralysing action upon the motor nerves, death resulting from asphyxia caused by paralysis of the muscles of respiration. It is possible that curare contains two active alkaloids, one having a paralysing and the other a tetanising action. It has been stated that curare does not act as a poison when taken into the stomach, but only when administered hypodermically. What is more probably the case is that a dose can be taken by the stomach without injury, which would, if given subcutaneously, produce violent symptoms, and even death. The explanation of this is probably found in the ready excretion of curare through the kidneys; if taken by the stomach, only small quantities of it pass from the stomach into the circulation at a time, whereas if subcutaneously injected the whole of the poison is at once introduced into the circulation, and so produces its effect upon the nervous system. The active principle of curare is an alkaloid curarine. With strong nitric acid curarine gives a deep red or purple colour. With concentrated sulphuric acid it gives a lasting deep blue or violet colour. Curarine can be distinguished from strychnine by the colour that it gives with nitric acid or with sulphuric acid, and also by the fact that potassium bichromate produces an amorphous precipitate of curarine chromate, whereas strychnine chromate is a crystalline precipitate.

Savin

Savin (Plate XII.) consists of the leaves and tops of *Juniperus Sabina* (Nat. Ord. Coniferæ). It contains a yellow volatile oil, the oil of savin. Both the powdered tops and the oil are powerful irritants. They are supposed by ignorant persons to possess powerful ecbolic properties, and are frequently taken with the object of procuring abortion, in the form of powdered savin, or as a decoction or infusion of the savin tops, or as the

Plate XL.

oil. The use of savin, or its oil, generally results in the illness or death of the woman, without abortion having been effected. If uterine contractions are set up by savin, and the contents of the uterus expelled, the result is brought about by the powerful irritant effect of the savin upon the stomach and intestines, causing a reflex contraction of the uterus from stimulation of the sympathetic nerve-endings. In such a case the expulsion of the contents of the uterus is almost certainly followed by the death of the woman, and death is generally preceded by hæmorrhage. Savin is a drug which should not be given to pregnant women. In the case of *Reg.* v. *Billingham and Nightingale* (Staffs. Ass., 1893), the two prisoners were convicted of the administration of pills to a young woman with the object of procuring abortion. The pills were analysed by Bostock Hill and the author, and were found to contain oil of savin and dragon's blood. Their administration produced in the woman pain, vomiting, and diarrhœa, but did not procure abortion.

Symptoms.—The symptoms of poisoning by savin are mainly those of gastro-intestinal irritation, and consist of a burning sensation in the throat and stomach, vomiting, diarrhœa, and strangury. Collapse, laboured respiration, and coma occasionally occur.

Post-mortem appearances.—Portions of the leaves or powdered stems may be discovered in the contents of the stomach or intestines. If a portion of the crushed or powdered stem be found, it should be examined under the microscope for the disc-bearing wood cells that are found in the wood of coniferous plants. As a rule, inflammation of the mucous membrane of the stomach and intestines is found, but it is not invariably present.

Tests for oil of savin.—I. The peculiar terebinthinate odour.

II. With strong sulphuric acid it gives a dark brown colour. On diluting the coloured liquid with distilled water, a dense white precipitate settles. This latter portion of the test is

useful as a confirmatory reaction, but is not to be relied upon as being diagnostic of the presence of oil of savin.

Male Fern

The rhizome of *Aspidium Filix Mas*, the *male fern*, is the part of the plant used. The active principle is filicic acid. In poisonous doses male fern is a convulsant, producing afterwards general paralysis and paralysis of the heart. The oil of male fern, or liquid extract of male fern, as it is called in the British Pharmacopœia, is obtained by extracting male fern with ether, and then removing the ether by distillation. It is used as an anthelmintic, especially for tape-worms. Dixon Mann [1] mentions the case of a man who took in two doses an ounce and a half of the extract of male fern, which was given in mistake for a drachm and a half. Soon after the first dose he felt unwell, and after the second, which was given some hours later, vomiting and purging came on. These symptoms were quickly followed by cramps, profuse sweating, delirium, and coma, which ended in death in about twenty hours after the drug was taken. A case [2] is also related of a child, aged two years and three-quarters, who took eight capsules—each containing about fifteen grains of extract of male fern mixed with the same quantity of castor oil—in the course of five hours. She became somnolent, paralysed, and died after the occurrence of some spasms. The mucous membrane of the stomach in this case showed petechial ecchymoses; there was also well-marked injection of the mucous membrane of the intestines. The peculiar point in this case is that three weeks previously the child took double the quantity of the extract, but without the castor oil, which points to the advisability of the administration of some aperient other than castor oil, if the extract of male fern be given.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube, or an

[1] *Forensic Med. and Toxicol.* [2] *Ibid.*

emetic of mustard and water should be given. Stimulants should be administered to counteract the depressing effect of the poison.

Santonin

Santonin is a glucoside obtained from *Santonica*, which consists of the unexpanded flower-heads of *Artemisia maritima* and other species of *Artemisia*. Santonin is employed as an anthelmintic for round-worms and thread-worms, and as it is used chiefly among children, cases of poisoning with santonin are more likely to occur with them than with adults.

Symptoms.—(i) The special symptom is a peculiar disturbance of vision, objects appearing at first blue, then yellow; finally colour-blindness results for some time. This curious fact is explained by Hufner and Helmholtz as being due to the action of the santonin on the violet-seeing elements of the retina, which are first excited, producing the blue vision, and then paralysed, producing the yellow vision. (ii) Headache, giddiness, and a species of intoxication frequently occur. (iii) Gastric pain, vomiting, and laboured respiration may be produced. (iv) In large doses convulsions, stupor, loss of consciousness, and death from collapse occur. (v) The urinary secretion is increased, and the urine is coloured saffron-yellow.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube; if neither of these be procurable, an emetic of mustard and water should be given. Stimulants should be administered to counteract the depressing action of the poison, and if convulsions are present, they should be treated with potassium bromide and chloral.

Fatal dose.—About two grains of santonin have proved fatal to a child, aged five and a half years, in twelve hours. Recovery, in a child, has followed after taking ten grains. Recovery has taken place, in a man, after taking an ounce of santonin in mistake for Epsom salts.

Analysis and tests.—Santonin can be extracted from acid solutions by means of chloroform. To the residue left, on

evaporation of the chloroform extract, the following tests may be applied :—

I. Strong sulphuric acid is diluted with half its bulk of water, a little of the diluted acid is added to some santonin in a porcelain dish, and the mixture warmed on a water-bath until a yellow colour is developed; while warm a few drops of dilute solution of ferric chloride are added drop by drop; as each drop falls in, a ring of a beautiful red or reddish-violet colour is produced around each drop, changing to purple, and afterwards to brown.

II. Santonin dissolves in a solution of caustic potash in absolute alcohol, producing a carmine-red colour which soon fades.

III. The saffron-yellow colour given to the urine by santonin may be confused with a similar colour produced in the urine by persons who have been taking rhubarb. If caustic soda be added to the urine coloured with santonin or rhubarb, a red colour is produced in either case; but, if the urine so reddened be digested with zinc dust, the colour of the santonin urine fades, while that of the rhubarb urine remains. The reddened urines may also be distinguished by adding an excess of slaked lime stirred with water, and filtering, when the filtrate from the urine reddened with rhubarb will be found colourless, while that from the urine reddened by santonin retains its colour.

CHAPTER XX

Poisoning by elaterium—Croton oil—Oil of turpentine – Oil of pennyroyal —Camphor– Poisonous fungi—Distinction between poisonous and edible fungi—Ergot—Acute and chronic ergot poisoning.

Elaterium

Elaterium is a sediment from the juice of the *squirting cucumber*, *Echalium Officinarum* or *Echalium Elaterium*. In poisonous doses it is a severe gastro-intestinal irritant, producing griping and vomiting, accompanied by considerable depression, salivation, and spasms. One-sixth of a grain of elaterium has caused the death of a woman aged seventy; two-fifths of a grain has also proved fatal. Recovery has taken place after three-quarters of a grain of elaterin, which is the active principle of elaterium, has been taken.

Croton Oil

This is the fixed oil expressed from the seeds of *Croton Tiglium*. Both the seeds and the oil are very poisonous. Croton oil is used in medicine, in doses of one or two drops, as a powerful purgative. In larger doses it acts as a strong cathartic, producing inflammation of the mucous membrane of the stomach and intestines. It also produces inflammation of the skin if applied to it. Its poisonous properties are dependent upon crotonic acid, a fatty acid existing in the oil.

Symptoms.—If poisonous doses of croton oil are taken, a burning sensation, accompanied by pain, is felt in the mouth, throat, and stomach, followed by violent vomiting and purging,

and considerable collapse. Death may take place in a few hours. Death has taken place after twenty drops and thirty drops of the oil have respectively been taken. In another case, mentioned by Taylor, a man swallowed by mistake two drachms and a half of the oil; death occurred in four hours. In another case half an ounce of the oil was taken by mistake, and produced violent vomiting, purging, and symptoms of collapse, but recovery took place at the end of fourteen days. Four of the seeds have caused death.

Oil of Turpentine

Symptoms.—If poisonous doses of oil of turpentine are swallowed, the following symptoms are generally produced:—(i) Burning pain in the mouth and throat, followed by vomiting and diarrhœa. (ii) Coldness of the surface, and a condition resembling that of alcoholic intoxication. (iii) Strangury and hæmaturia; the urine passed possesses a violet-like odour. (iv) Spasms, and coma in fatal cases.

Chronic turpentine poisoning may arise from persons sleeping in newly painted rooms, or from being exposed to the vapour of turpentine in industrial occupations. The symptoms consist of dizziness, depression, painful micturition, hæmaturia, albuminuria, and a violet-like odour of the urine.

Treatment.—In cases of acute poisoning, the stomach should be emptied and washed out by means of the stomach-pump or stomach-tube; if neither of these be procurable, an emetic of mustard and water should be administered. Demulcent drinks should be freely given, and opium or morphine administered, if necessary, to counteract the pain.

Post-mortem appearances.—Small hæmorrhages have been found in the mucous membrane of the stomach, and erosion of the membrane has also occurred. The blood is generally dark coloured.

Fatal dose.—An infant, five months old, has died from the administration of a tablespoonful of oil of turpentine. In

another case an infant recovered after the administration of four ounces. Six ounces have proved fatal to an adult.

Oil of Pennyroyal

Oil of pennyroyal has the popular reputation of being an emmenagogue and ecbolic. In reference to this, Taylor states that it has neither emmenagogue nor ecbolic properties, but this statement probably requires some modification. Marshall [1] relates a case of abortion, following the taking of three drachms of the essence of pennyroyal, in which severe flooding and expulsion of the ovum were produced, followed by collapse of the patient. He states that he has been informed by married women of considerable intelligence and powers of observation, that they are absolutely certain that pennyroyal will bring on the catamenia when they have been suppressed. As pennyroyal is not employed by medical practitioners, there are obviously great difficulties in collecting evidence as to the real action of such a drug. Flynn [2] relates a case of a woman, two months pregnant, who took three fluid drachms of essence of pennyroyal with the object of procuring abortion. When seen an hour later she was very excited, the pupils were considerably dilated, and the pulse at the wrist was very feeble, and a few minutes later was imperceptible. After treatment with emetics she rapidly recovered..

Camphor

In poisonous doses camphor acts as an irritant and depressant to the nervous centres, and produces convulsions. The symptoms generally consist of pain in the stomach, headache, vomiting, strangury, occasionally delirium or collapse, with pallor and lividity of the face, laboured breathing, and cold skin ; coma may supervene. Taylor mentions the case of a woman who took twenty grains of camphor, and in whom

[1] *Brit. Med. Jour.*, 1890. [2] *Ibid.*, 1893.

serious symptoms occurred, but recovery eventually took place. Blyth mentions a case of a woman, pregnant four months, who took about 184 grains of camphor with the object of procuring abortion; she suffered from severe symptoms, and at the end of about six days aborted, and died in a comatose state. Davies[1] mentions a fatal case of camphor poisoning, which occurred in a child, aged two years and eight months, after eating a piece of solid camphor weighing about thirty grains. Prostration, very rapid pulse, and severe convulsions followed. Vomiting was induced, and the stomach subsequently washed out, but convulsions continued, and finally ended in death eighteen hours after the camphor had been taken. The presence of camphor in the stomach is at once detected by its odour.

Poisonous Fungi

Certain fungi possess noxious and even fatal properties. The poisonous properties of fungi, however, depend very much on the idiosyncrasy of the individual, since the same fungi have produced in different members of the same family different effects; in some cases the symptoms of narcotic poisoning result, whilst in others the symptoms of irritant poisoning are produced. In the following table the principal non-poisonous and poisonous fungi are enumerated:—

Non-poisonous fungi, or mushrooms	**Poisonous fungi** (Toxic principle, phallin)	**Poisonous fungi** (Toxic principle, muscarine)
Agaricus campestris	Agaricus phalloides	Agaricus muscarius
,, oreades	,, mappa	,, pantherinus
Lepiota procera	,, verna	Boletus luridus
	,, recutita	
	,, porphyria	

The most important fungus as a poison is agaricus phalloides. Falck collected fifty-three cases of poisoning by it,

[1] *Brit. Med. Jour.*, 1887.

of which forty were fatal. The cause of this fatality is partly that it is mistaken for the common mushroom, to which it bears a slight resemblance, and partly that its toxic properties do not manifest themselves until after absorption of the poisonous principle into the blood, as its action is that of a blood poison and not that of a gastro-intestinal irritant. Agaricus phalloides is a very common autumn fungus, and has frequently been mistaken for mushrooms. The principal fungi eaten in England as mushrooms are the agaricus campestris and the agaricus oreades, or the champignon. The distinctions between the poisonous agaricus phalloides and the mushroom are the following:—

Agaricus phalloides	**Agaricus campestris** (the common mushroom)
1. Permanent white gills	1. Gills pink in young mushrooms, black in old ones, and of various shades of chocolate-brown between the two, according to age
2. Stem hollow	2. Stem solid
3. Large bulbous base	3. Base not bulbous

Kobert has found that the poisonous principle of agaricus phalloides is a vegetable toxalbumin, which he calls phallin, and which is present to the extent of one per cent. in the dried fungus. The action of phallin is to destroy the red blood corpuscles, causing the serum to become reddish, and the urine blood-stained. By the dissolution of the blood corpuscles fibrine is liberated, and, in consequence, thromboses are liable to form. These are especially apt to occur, along with multiple ecchymoses and fatty degeneration, in the liver, and Kobert points out the similarity between the affection of the liver by this poison, and the condition of the liver in cases of phosphorus poisoning, and in acute yellow atrophy of the liver. Phallin also occurs in, and is the toxic principle of, agaricus mappa, agaricus verna, agaricus recutita, and agaricus porphyria.

Agaricus muscarius, agaricus pantherinus, and boletus luridus constitute another group of poisonous fungi, the poisonous principle of which is the alkaloid muscarine. Agaricus muscarius is the fly-fungus, so-called because a decoction of it has been employed as a poison for flies. With these three fungi symptoms often occur within an hour, or an hour and a half, and death may ensue in from six to ten hours. Muscarine, according to Brunton, produces uneasiness in the stomach, vomiting, purging, dyspnœa, vertigo, faintness, prostration, and stupor. It slows the heart by stimulating the inhibitory cardiac mechanism, lowers blood-pressure, depresses the respiratory centres, causes contraction of the pupils, and stimulates the secretion of sweat and saliva. Muscarine strongly resembles pilocarpine in its action, but only causes contraction of the pupils when administered internally; if applied locally it dilates the pupils, whereas pilocarpine causes contraction of them, whether administered internally or locally. Muscarine is antagonistic to atropine. There is another alkaloid in agaricus muscarius called pilzatropine, which is a form of atropine, and which modifies the action of muscarine, so that, in some cases of poisoning by this fungus, contraction of the pupil and salivation may be absent. A third alkaloid, named amanitin, is also present in agaricus muscarius, and according to Kobert is present in most fungi, both edible and poisonous. Of itself it is practically inert, but it is apt—as, for instance, by the incipient decay of the fungus—to become converted into lecithin and neurin, and thus to give rise to gastro-enteritis. Brieger states that it is often to be found after death in the bile as cholin, and in the other tissues as neurin. This is the explanation of the symptoms of poisoning which are sometimes caused by the common mushroom and other edible fungi.

Helvellic acid, which occurs in Helvella esculenta, is removed by water, so that the broth in which the helvellæ have been cooked is more poisonous than the fungi themselves. Its action resembles that of phallin, and causes an analogous train of

symptoms, owing to dissolution of the blood corpuscles. Cases of poisoning occasionally occur in Great Britain from the small black-spored agarics, such as agaricus campanulatus, agaricus semiglobatus, and agaricus semilanceolatus. They are seldom fatal, the symptoms principally consisting of giddiness and delirium, which occur soon after the fungus is eaten, and, if vomiting be induced, soon pass away.

Symptoms.—The following are the symptoms produced by poisoning by the more important poisonous fungi. As a rule, the symptoms consist of both the narcotic or neurotic and irritant types, but the predominance of the symptoms of one or other type is very much subject to the idiosyncrasy of the individual affected. The symptoms may occasionally come on within one hour, but may be delayed for six, ten, or more hours. (i) Abdominal pain, thirst, nausea, vomiting, and purging. (ii) Collapse, with cold sweats and cyanosis. (iii) Small pulse and laboured respiration. (iv) Giddiness, trembling, dilated or contracted pupils, illusions, delirium, stupor, and coma. (v) Convulsions may occur, especially if a fatal issue be impending. (vi) Hæmoglobinuria, hæmaturia, and albuminuria may occur, or occasionally the urine may be suppressed. Very occasionally jaundice occurs.

Treatment and antidotes.—The stomach should be emptied by means of an emetic of mustard and water, or by the hypodermic administration of apomorphine. The bowels should be cleared, by a castor oil purge, of any particles of fungi that may have passed into them. Stimulants and external warmth should be employed to counteract the collapse, and atropine should be hypodermically injected in doses of $\frac{1}{50}$ to $\frac{1}{30}$ of a grain, if the pupils are contracted, to counteract the poisonous effects of the muscarine. Morphine should be administered if there be much pain.

Post-mortem appearances.—Careful search should be made in the contents of the stomach and intestines for portions of fungi. Inflammation of the mucous membrane of the stomach and intestines is generally present. Multiple ecchymoses of

the gastro-intestinal mucous membrane, and in the liver, are frequently found, together with localised gangrenous spots on the mucous membrane of the stomach and intestines. The heart and liver are frequently in a state of fatty degeneration, and the blood is generally fluid. The changes in the liver may closely resemble those found in cases of phosphorus poisoning.

Fatal period.—Death has occurred at the following different periods in various cases of poisoning by fungi: twenty-nine hours, forty-eight hours, three days, and fourteen days after the first appearance of symptoms.

Ergot

Ergot consists of the compact mycelium of a fungus named *Claviceps Purpurea*, which grows on the seeds or grains of the common rye. Ergot is an ecbolic. It is certainly capable of causing contractions of the uterus during the later stages of pregnancy, but in the earlier stages it is considered somewhat doubtful whether it can initiate contractions of the uterus or produce them with sufficient force to cause the expulsion of the uterine contents. Some authors state that ergot does not act unless the natural contractions of the uterus have already commenced; but since, during pregnancy, uterine contractions normally occur, it is quite conceivable that ergot may be able to increase the force of these contractions, although in the earlier months of pregnancy it may not be able to augment them sufficiently to procure abortion. It is certain that it frequently fails to bring about a miscarriage, although very large and repeated doses have been tried. In a case of suspected criminal abortion, investigated by the author several years ago, a large quantity of the liquid extract of ergot was found in the stomach; death had resulted from uterine hæmorrhage, but the fœtus (fourth or fifth month) was still within the uterus undetached. The chemical composition of ergot has been the subject of numerous investigations, but none of the principles isolated can be said to represent the full activity of the drug. Ergot

certainly contains more than one active principle; those known at the present time are ergotinic acid, sphacelinic acid, and an alkaloid cornutine; ergotin is a mixture of these, and possibly of other constituents. Poisoning by ergot may be acute or chronic, the symptoms being different in the two kinds of poisoning.

Symptoms of acute ergot poisoning.—(i) Epigastric pain, thirst, nausea, and vomiting. (ii) Giddiness, cardiac oppression, numbness, and tingling of the extremities. (iii) Shivering, coldness, cramps, delirium, coma and convulsions.

Davidson [1] records a case of a female, aged twenty-eight, who took ergot, and in whom the symptoms consisted mainly of vomiting of blood, passing of bloody urine, intense jaundice, and stupor. This case is a somewhat exceptional one, as jaundice and vomiting of blood have not generally been noticed.

Treatment and antidotes.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube; if neither of these be available, an emetic of mustard and water should be given, or a hypodermic injection of apomorphine; stimulants and external warmth should be employed. An inhalation of three minims of nitrite of amyl, or the administration by the mouth of $\frac{1}{50}$ of a grain of nitroglycerine in solution, is advisable to counteract the cardiac oppression; either of these remedies should be repeated if necessary.

Post-mortem appearances.—Ecchymoses and extravasations of blood are generally found on the surface of, and within, the internal organs. Fatty changes may be found in the liver and kidneys.

Chronic Ergot Poisoning

This occurs from eating bread made with grain upon which the ergot fungus has grown. Epidemics of chronic ergot poisoning have occurred from time to time, especially in Russia and Germany. The first symptoms are those of gastro-enteritis, accompanied by nervous symptoms, such as lassitude, depres-

[1] *The Lancet*, 1882.

sion and dizziness. Later on the symptoms may assume the gangrenous form, or the spasmodic or convulsive form, or both. These forms of ergotism are, no doubt, due to the action of the ergot upon the muscular walls of the arterioles, producing sufficient contraction or spasm to shut off the supply of blood to the parts to which they are distributed.

The gangrenous form of ergotism.—In this form patches of anæsthesia first occur, or acute pain may be felt in the limb or limbs about to be affected, which symptoms are generally accompanied by an erythematous or erysipelatous condition of the skin. Gangrene then sets in, and is generally of the dry type, but occasionally moist gangrene occurs; the toes and fingers are the parts most frequently affected. The gangrenous part separates from the healthy by a slow moist process of ulceration, and in this way the toes, fingers, and even the legs, may come off. In very exceptional cases the skin only may be attacked by gangrene.

The spasmodic or convulsive form of ergotism.—This generally commences with either anæsthesia of the fingers, toes and extremities, or by creeping sensations spreading from the toes and fingers along the limbs. These symptoms are followed by muscular spasms of the flexors of the fingers and toes, and of the extensors of the foot. Occasionally this muscular spasm extends to the back, producing opisthotonos. The spasms are intermittent, lasting from a few minutes to some hours, and during their occurrence are extremely painful. Symptoms of cerebral disturbance also frequently occur, such as flashes of light before the eyes, noises in the ears, giddiness, lightning pains, hallucinations, delirium, and stupor.

Fatal dose.—Nothing definite is known of the actual amount of ergot required to produce a fatal result. Poisonous symptoms have been produced by doses of from half a drachm to two drachms of ergot in powder.

Analysis and tests.—The detection of ergot in the tissues is impossible. From the contents of the stomach, or from ergotised grain or bread, an extract may be made by means of

hot alcohol acidulated with sulphuric acid. This extract is red in colour, and if examined with a spectroscope shows two bands, one in the green and another in the blue, the latter being broader and more distinct. If powdered ergot, or the residue left by the evaporation of the tincture or liquid extract, be rubbed with solution of caustic potash and gently heated, it acquires a reddish colour, and evolves a peculiar fishy odour of trimethylamine or of propylamine. This colour reaction is only a pigment change, and is therefore only produced if a portion of the colouring matter of the ergot be present; it may not, therefore, be produced by the residue left on evaporation of the tincture of ergot, as the pigment is insoluble in alcohol.

VARIOUS ORGANIC POISONS

CHAPTER XXI

Poisoning by petroleum—Paraffin oil—Benzene—Nitrobenzene—Dinitro benzene — Aniline — Antifebrin — Exalgin—Antipyrin—Naphthaline—Carbolic acid—Picric acid—Resorcin—Pyrogallic acid—Salicylic acid —Salol.

Petroleum and Paraffin Oil

Petroleum is found naturally in certain parts of the world. It consists of a mixture of the higher saturated hydrocarbons; the crude petroleum is purified by rectification, and is then free from colour, or possesses a pale yellowish colour in bulk, and a peculiar penetrating odour. Petroleum is largely used for illuminating purposes. The lighter and more volatile portions of the crude petroleum are known under the names of *cymogene*, *gasolene*, *naphtha*, and *benzoline*. Benzoline, which is also known as *mineral naphtha*, *petroleum naphtha*, *petroleum spirit*, and *petroleum ether*, is a very volatile and inflammable liquid, and consists of a mixture of the lighter hydrocarbons obtained by distillation from petroleum. Benzoline must not be confounded with benzol or benzene (see p. 357). Benzoline is the illuminant employed in sponge lamps.

Paraffin oil, known also as *kerosine* and *mineral oil*, is a mixture of the saturated hydrocarbons obtained by distillation of shale. Petroleum and paraffin oil are frequently sold indiscriminately for one another, and as regards their toxic effects, there is practically no difference between them. Neither of them, however, can be considered to be an active poison. In cases of poisoning by either of these substances the symptoms

generally are vomiting, giddiness, collapse, with pallor of the face, cold clammy skin, and weak pulse. Eructations occur, and there is a marked odour of the poison in the breath and in the vomited matters. Pain in the stomach and throat may or may not be felt. The vomited matters generally contain blood, and blood may be passed in the motions. On the other hand, a large quantity of paraffin oil may be swallowed without any vomiting or other sign of gastric irritation. Occasionally the urine contains albumen and blood. Several cases have been recorded in which half a pint of paraffin oil has been swallowed and recovery has taken place. A pint of petroleum has been taken, with the production of only temporary disorder.

BENZENE AND MEMBERS OF THE BENZENE SERIES

Benzene

Benzene is commonly known as *benzol,* and as *coal-tar naphtha.* It is one of the products of the dry or destructive distillation of coal, and is obtained from coal-tar by fractional distillation. Commercial benzene contains small quantities of other liquid hydrocarbons. The symptoms of acute poisoning somewhat resemble those of alcoholic poisoning, there being a stage of excitement, with flushed face, followed by collapse and a tendency to stupor or coma. In the stage of collapse the breathing is stertorous and the pulse weak. Vomiting may or may not occur. The odour of benzene is generally perceptible in the breath for many hours after the poison has been swallowed. Benzene produces gastro-enteritis, and the treatment should consist in washing out the stomach as soon as possible, as it is rapidly absorbed. Death has occurred in a child, two years old, in ten minutes after a mouthful of benzene was swallowed. A boy, aged twelve, died in less than three hours after swallowing about three ounces of benzene. Rosenthal [1] reports a case of

[1] *Centralbl. f. inn. Med.,* 1894.

poisoning in a girl, aged a year and a half, the quantity of benzene taken being uncertain. When seen ten to fifteen minutes afterwards, the child was in a condition of stupor, the pulse was small and frequent, and the respirations 60 to 70. The stomach was washed out, the washings smelling strongly of benzene and containing blood-stained masses of mucus. In about six hours the child had considerably improved, and subsequently recovered completely. Poisonous symptoms may also result from the inhalation of benzene vapour. Workmen engaged in distilling benzene have suffered from anæmia, accelerated pulse, and hot skin, and emit the odour of benzene in the breath; occasionally symptoms of a more serious nature are observed, such as intoxication, accompanied by delirium, epileptiform attacks, loss of sexual power, paralysis, and disturbance of sensation, which may take the form of anæsthesia or hyperæsthesia.

Benzene poisoning by inhalation has also been observed occasionally among glove-cleaners.

Tests.—I. The odour.

II.—By treatment with strong nitric acid nitrobenzene is formed, which can be recognised by its peculiar odour (see p. 360), and by its conversion into aniline when subjected to the action of nascent hydrogen (see p. 361).

Nitrobenzene

This body is also known as *nitrobenzol* and *essence of mirbane*. It is a yellow liquid, possessing a strong odour resembling that of the essential oil of bitter almonds, and is on this account sometimes known as *artificial oil of bitter almonds*. It is used as a substitute for the essential oil of bitter almonds in perfumed soaps and confectionery.

Symptoms.—These do not generally appear till from a quarter of an hour to two or three hours have elapsed after taking the poison. (i) Uneasiness, headache and vomiting may occur; the vomited matters possess the characteristic odour of

nitrobenzene. (ii) Cyanosis, which affects the mucous membrane of the lips, gums tongue, mouth, and pharynx, imparting a livid bluish hue. The nails are also coloured of a purple or dark blue colour, looking as if they had been stained with blackberry juice. The surface is cold and clammy. (iii) Unsteady gait, feeble quick pulse, shallow and irregular breathing, with the odour of the poison in the breath. (iv) Drowsiness, stupor, and coma occasionally supervene ; convulsions may occur at an uncertain interval after the stupor.

Hodson [1] records the case of a pharmacist, aged forty-eight, who, by mistake, drank two drachms of nitrobenzene. An hour and a half later he became giddy, but was able to walk a distance of about half a mile, feeling all the time as though intoxicated. He then became partly unconscious, and when seen three hours and a half after taking the poison he was insensible, collapsed, and extremely cyanosed. Five hours after taking the poison, the patient having been treated in the meantime, consciousness began to return, and he then gradually improved, but the cyanosis did not disappear until the eighth day. Severe symptoms, and even death, may result from the absorption of nitrobenzene into the system in the form of vapour. Thompson [2] records the case of a workman who for several hours was engaged in pouring nitrobenzene from large drums into smaller vessels, and who was seized with the following symptoms :—dizziness, headache, vomiting, hurried respiration, the breath smelling of nitrobenzene. The surface of the skin was of a dark purple hue, especially marked on the mucous membrane of the lips and beneath the finger nails. The heart's action was rapid, and there was some tendency to coma when at rest; the urine was very dark in colour, and smelt strongly of nitrobenzene. The treatment was simply expectant, and was directed to keeping the patient alive while assisting the lungs and the kidneys in the elimination of the poison. Five days later the patient had recovered, but still retained a peculiar complexion. Letheby has recorded the case

[1] *The Lancet*, 1891. [2] *Brit. Med. Jour.*, 1891.

of a man, aged forty-two, who spilt some nitrobenzene over his clothes, and went about for several hours breathing an atmosphere charged with the vapour of the poison. He subsequently became drowsy, unsteady in his gait, and presented the appearances of intoxication; stupor came on and deepened into coma, and the man died about nine hours after the commencement of symptoms.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube. Stimulants and external warmth should be employed, and artificial respiration resorted to if necessary.

Post-mortem appearances.—The characteristic odour of nitrobenzene is recognised on opening the cavities of the body. The blood is chocolate-brown or dark in colour.

Fatal dose.—Twenty drops have proved fatal, but recovery has occurred after very nearly an ounce of the poison has been swallowed, owing to the quick employment of rapid and efficient treatment. Alcoholic liquids, on account of their solvent power on nitrobenzene, hasten and intensify its action, so that a person swallowing nitrobenzene after taking spirits is likely to be more affected than if the spirits had not been taken. Stevenson [1] records a case in which a young man, aged twenty-one, took on sugar seven doses of nitrobenzene, each of twenty minims, spread over a period of about fifty hours. Six hours after taking the last dose he became insensible, and the body assumed a pale blue colour. With suitable treatment he recovered. From some of his urine nitrobenzene was extracted by shaking with chloroform.

Fatal period.—From one to twenty-four hours.

Analysis and tests.—Nitrobenzene may be separated from organic substances by distillation in the presence of sulphuric acid. To the distillate so obtained the following tests may be applied.

I. The smell, which closely resembles that of the essential oil of bitter almonds. It may be distinguished from the latter

[1] *Guy's Hosp. Repts.*, 1876.

by adding a drop of strong sulphuric acid to a few drops of the liquid in a porcelain evaporating dish. The nitrobenzene is not affected, whereas the oil of bitter almonds acquires a rich crimson colour with a yellow border.

II. By adding some of the nitrobenzene to zinc and hydrochloric acid, or to iron filings and dilute sulphuric acid, aniline is produced by the reducing action of the nascent hydrogen on the nitrobenzene. The liquid is filtered from the undissolved zinc or iron, when the filtrate will contain the aniline in solution as a soluble salt. The solution may be divided into three parts and the following tests for aniline applied.

(*a*) The addition of bleaching powder produces a purple or violet colour.

(*b*) On addition of solution of alcoholic caustic potash and a few drops of chloroform the disagreeable odour of phenyl isocyanide is produced on shaking and warming the liquid.

(*c*) If the solution be rendered alkaline with caustic potash, the aniline can be extracted by shaking with ether. After removal and evaporation of the ether, the aniline is mixed with water, and on the addition of a watery solution of carbolic acid and sodium hypochlorite, a dark blue colour is produced, which changes to red on the addition of hydrochloric acid.

Dinitrobenzene

This substance is a solid of a yellow or yellowish-brown colour. It enters into the composition of *roburite, bellite* and *sicherheit*, explosives used in coal mines for the purpose of blasting. Roburite generally consists of a mixture of chlorodinitrobenzene and ammonium nitrate. Poisoning by dinitrobenzene occurs in factories where it is used, and is due to either fine particles of it floating in the air, or to the vapour of it passing into the system. Probably prolonged handling of the substance may also lead to its absorption through the skin. The symptoms are similar to those produced by nitrobenzene.

Chronic poisoning.—Chronic poisoning by dinitrobenzene,

which occurs amongst those engaged in preparing or handling it, produces the following symptoms:—Headache, loss of appetite, pain in the stomach, and a feeling of lassitude. The lips acquire a bluish colour and the skin and conjunctivæ a yellow tint. The urine is dark in colour from the presence of dinitrobenzene or one of its derivatives. Symptoms of peripheral neuritis, such as numbness, painful sensations of the nerve-endings, and cramps may supervene.

Snell[1] records several cases of chronic poisoning by dinitrobenzene among workers engaged in the manufacture of certain explosives, such as roburite and sicherheit. He finds that the dinitrobenzene acts as a poison whether ingested, absorbed by the skin, or inhaled into the lungs in the form of vapour or dust. The symptoms of chronic poisoning as described by him are—pallor of the face, bluish colouration of the lips, fingers, toes, and of the nails of the fingers and toes, nausea and vomiting occasionally, weakness, giddiness and staggering. Snell also finds that amblyopia is a common symptom, accompanied by concentric contraction of the visual field, and, in many cases, by a central colour scotoma; there is also some blurring of the edges of the optic disc and a varying degree of pallor of its surface. He also found that the action of the poison on the blood is peculiar and definite. Blood drawn from the fingers of affected persons was found to be thin and black-looking, and to contain large coloured corpuscles (megalocytes), while the ordinary red corpuscles were smaller than normal. The appearances were like those seen in pernicious anæmia. The urine was of a brown or blackish colour; this colour was not due to blood or bile, but to some pigment belonging to the aromatic series. The blue asphyxiated appearance of the patients was probably due to the production of this or some other pigment conveyed in the blood.

Poisoning may occur from the fumes of chloronitrobenzene given off by unexploded roburite, the symptoms being similar to those of dinitrobenzene. Spurgin[2] relates a peculiar case of

[1] *Brit. Med. Jour.*, 1894. [2] *Ibid.*, 1891.

such poisoning. A boy aged sixteen had slept for one night in a bedroom, the floor of which had been sprinkled with roburite to clear out cockroaches; the next morning the boy was cyanosed, the nails were of a blue-black hue, the tongue, lips and mouth nearly black, and the body and face were livid. He was collapsed, the temperature was subnormal, the pulse very weak and rapid, and the breathing laboured. The treatment consisted in the removal of the boy from the room and the employment of stimulants and warmth externally; after three days the patient had nearly recovered.

Treatment.—The same as for nitrobenzene poisoning (see p. 360). Chronic poisoning requires removal from the influence of the poison.

Post-mortem appearances.—The blood has been found chocolate or dark in colour, and ecchymoses have been found in the various mucous membranes.

Aniline

Aniline, or phenylamine, is a colourless liquid gradually changing to a brownish colour by exposure to air. The various aniline dyes are obtained by the oxidation of aniline with oxidising agents of different degrees of strength.

Symptoms.—In cases of aniline poisoning the symptoms come on in from five minutes to an hour or more after swallowing the poison, and are—(i) Nausea and vomiting. (ii) Cyanosis, the lips, gums, tongue, and nails assuming a purple or bluish hue. The skin is cold and clammy. (iii) Laboured respiration; the odour of the aniline is frequently noticeable in the breath. (iv) Giddiness, drowsiness, coma. Similar symptoms may be produced by the inhalation of the vapour. The cyanosis produced by aniline and nitrobenzene compounds is probably not that of asphyxia, but is due to the formation of coloured aniline products formed in the system, since the purple or bluish colour may occur without any sign of dypsnœa, and, even if dyspnœa has been present,

the blue colour remains for some time after the breathing has become normal.

Treatment.—The same as for nitrobenzene poisoning (see p. 360).

Post-mortem appearances.—The blood has been found of a chocolate colour.

Fatal dose.—Six fluid drachms have proved fatal.

Analysis and tests.—Aniline may be separated from organic matter by rendering it alkaline with caustic potash and then distilling. To the distillate the various tests for aniline mentioned in connection with nitrobenzene may be applied (see p. 361).

Aniline Colours

Very few of the aniline colours are injurious, and those few which are so are commonly toxic only in very large doses. This is a matter of considerable importance, as the aniline colours are widely used in articles of food. Leffmann states that an ounce of auramine will colour 2,000 lbs. of sugar candy a beautiful yellow colour; it is therefore difficult to believe, even should auramine be proved poisonous, that any child could eat sufficient sugar candy, dyed yellow with it, to produce any appreciable effect. The bad name that formerly attached to some of the red aniline dyes seems to have been due to impurities, such as arsenic, introduced in the process of manufacture. These dyes are, however, now prepared quite free from arsenic.

Antifebrin

This substance, which has acquired considerable use as an antipyretic, is also known as acetanilide and phenyl-acetamide. It occurs in small white odourless crystals, almost insoluble in cold water, but freely soluble in alcohol. If taken in poisonous doses, it produces symptoms of the aniline type.

Symptoms.—(i) Feeling of lassitude, impairment of sight, vertigo, sleepiness and unconsciousness. (ii) Collapse, cyanosis, and cold surface. (iii) Lowered respiration and pulse.

Warfvinge [1] relates a case of a man, aged thirty-five, who took two drachms of antifebrin. In five minutes he felt fatigued, and black and red clouds appeared before his eyes. Lassitude in the extremities came on, and he went to bed and slept for an hour and a quarter. On attempting to rise he became giddy, fell down unconscious, and sank into a comatose condition, with well-marked cyanosis, coldness of the hands and feet, decreased temperature, and a lowering of respiration and pulse. He continued in this comatose state sixteen hours, but recovered under treatment with hypodermic injections of camphor and ether. Cyanosis continued till the third day. The urine was deeply coloured and contained indican, but no blood, albumen, or sugar. Hartge has recorded a case in which a student took about 340 grains of antifebrin in addition to a considerable quantity of spirit. In this case there was no loss of consciousness; the respirations were rapid, the pulse accelerated, and the patient complained of inability to sleep. There was a marked blueness of the skin, which was general, but especially dark in the eyelids, chin, and temporal regions. There was no albuminuria or hæmoglobinuria. The blood corpuscles presented their normal appearance and character, though the colour of the blood was changed to a dark blue, as in aniline poisoning. On the third day the patient had recovered, and the blue colour had entirely vanished.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube; if neither of these be available, an emetic of mustard and water should be administered, or a hypodermic injection of apomorphine should be employed; stimulants and external warmth should also be used.

Severe symptoms have been produced (as in the case previously described) by an adult taking two drachms of antifebrin, and in an infant five months old by three to four grains. The blood has been found to contain methæmoglobin.

[1] *Hygcia*, 1892.

Analysis and tests.—Antifebrin may be extracted from acid solution by agitation with ether or chloroform.

I. Antifebrin behaves like aniline on warming with alcoholic caustic potash and chloroform, the disagreeable odour of phenyl isocyanide being produced.

II. If potassium bichromate be dissolved in strong sulphuric acid, and a drop of the mixture added to a fragment of antifebrin, a red colour is produced changing to brown and finally to dirty green.

III. If a fragment of potassium or sodium nitrite and a drop of strong hydrochloric acid are added to a fragment of antifebrin, a yellow colour is produced, which on warming changes to green and blue. On evaporating the liquid to dryness an orange residue is obtained, which on the addition of ammonia changes to red.

Exalgin

Exalgin or methyl-acetanilide is a substance possessing analgesic properties, and is used in the treatment of neuralgia, migraine, &c. In over-doses it exerts toxic effects, the symptoms being those of the aniline type. Reynery[1] records the case of a boy, two years old, who by mistake was given two powders, each containing five grains of exalgin. The first dose was vomited up about half an hour after being taken, and was succeeded by prostration and more vomiting. The second dose was given three hours after the first; an hour later the child's face and hands were of a dark blue tint, and the colour was still more marked in the nails, scrotum and penis. The lower lip and the tongue were blackened; there was copious vomiting, and free sweating of the head and upper part of the body; the pulse could not be counted. Under treatment with stimulants the patient recovered, but the discolouration of the skin and some of the other symptoms did not disappear till the end of ten days. Lloyd Jones[2] records the case of a woman, aged twenty-

[1] *Gaceta Medica. Municipal.*, 1892. [2] *Brit. Med. Jour.*, 1890.

four, who for five days was treated with two-grain doses of exalgin three times a day, but as the drug failed to give relief to the pain from which she was suffering, the doses were increased to four grains, and at the end of two days to six grains. After taking the six-grain doses three times a day for a week, the following symptoms of poisoning set in :—The lips and cheeks became blue, the pulse was small; nausea, giddiness, indistinctness of sight, and a feeling of weight at the epigastrium were complained of, and were followed by vomiting. The cyanosis continued to increase, and the nails, lips, and cheeks became deeply coloured. A few inhalations of nitrite of amyl were employed, and caused marked dilatation of the vessels, with considerable emphasis of the blueness, showing that the whole of the circulating blood was profoundly changed. Under treatment with stimulants, strychnine, and digitalis the patient recovered. Johnston [1] reports a case in which severe poisonous effects were caused in an adult man by three grains of exalgin.

Antipyrin

This is a rather complex benzene derivative, prepared from aniline, aceto-acetic ether, and methyl iodide. It is a white crystalline body, readily soluble in water, and is much used as an antipyretic and analgesic. Antipyrin is a powerful depressant, and in poisonous doses causes collapse, cyanosis, cold sweats and tumultuous heart beats. Rapin [2] describes a case of poisoning by antipyrin, which occurred in a lady, aged twenty-eight, suffering from severe sciatica. She took four doses of fifteen grains of the drug in the course of five days with good results, and without any unpleasant effects, but, after another dose of fifteen grains on the sixth day, she almost immediately felt an intense burning pain in the epigastrium, followed by vomiting and severe collapse. The cheeks and lips became almost black from cyanosis, and an itching rash came

[1] *Brit. Med. Jour.*, 1890.

[2] *Revue Méd. de la Suisse Romande*, 1888.

out all over the body. On the following day she had completely recovered. A somewhat similar case has been published by Whitehouse, in which the symptoms were caused by a seven-grain dose of antipyrin, and disappeared after hypodermic injection of atropine. Oscar Jennings relates a case in which 38·5 grains of antipyrine were given daily for a week for the relief of rheumatism. On the eighth day erythematous spots appeared on the arms, and on the ninth day the face was red, and the eyelids swollen. On the tenth day a rash came out all over the body, and there was catarrhal conjunctivitis, loss of appetite, singing in the ears, and collapse. Oscar Jennings states that the symptoms of poisoning are easily subdued by administering tincture of belladonna, or by subcutaneous injection of atropine. Sée, on the other hand, condemns the practice of giving belladonna or injections of atropine, as the symptoms of poisoning disappear spontaneously in from twenty-four to forty-eight hours if the drug be discontinued. Short[1] relates the case of a male adult, who, two hours after taking five grains of antipyrin, had flushing of the face, and, two hours later, suffered from redness and swelling of the nose and lips. A few hours later a crop of herpes-like vesicles appeared on the nose, lips, and inside of the cheeks ; the hands and feet were swollen, red, and itching, and the skin of the penis and scrotum and the anal margin were in a similar condition. The vesicles on the nose, lips, and mouth discharged profusely. The symptoms gradually subsided under treatment, and, at the end of a week, were followed by desquamation in the portions of the skin affected.

Analysis and tests.—Antipyrin can be extracted from an alkaline solution by means of chloroform. To the residue, left on evaporation of the chloroform extract, the following tests may be applied :—

I. If antipyrin be heated with strong nitric acid, and the liquid allowed to cool, a purple colour is produced ; on the

[1] *Brit. Med. Jour.*, 1892.

addition of water a violet precipitate is thrown down, and the filtered liquid will be of a purple-red colour.

II. A solution of antipyrin gives with ferric chloride a blood-red colouration, which is destroyed by the addition of a mineral acid. This reaction is given by urine containing antipyrin.

III. Antipyrin gives with nitrous acid a green colour. This test is best applied by dissolving potassium nitrite in water, adding excess of strong sulphuric acid, and then pouring into the mixture the solution of antipyrin. This test is common to the group of bodies of which antipyrin is a member.

Antifebrin can be distinguished from antipyrin by not giving a red colouration on the addition of ferric chloride.

Naphthalene

This is a hydrocarbon obtained as a bye-product in the preparation of coal gas. It is used as an antiseptic. In large doses it has produced toxic effects. Rossbach [1] relates a case in which a dose of ninety-three grains of naphthalene produced cyanosis, muscular twitchings, and dark-coloured urine. The colour of the latter was probably due to hæmoglobinuria.

Carbolic Acid or Phenol

This body is also known as *coal-tar, creasote,* and as *phenic acid.* It is contained in coal-tar, from which it is obtained, for commercial purposes, by fractional distillation. In the pure state carbolic acid is a colourless crystalline body, but it soon assumes a pink colour, due to the absorption of carbon dioxide from the air and the consequent formation of a small amount of aurin. It is slightly soluble in water, and readily soluble in alcohol, ether, and glycerine. If carbolic acid be mixed with one-tenth of its weight of water, it liquefies, forming *liquefied carbolic acid.* It possesses a characteristic odour and taste somewhat resembling creasote. It is a powerful antiseptic, and

[1] *Berliner Klin. Wochenschr.*, 1884.

a corrosive and irritant poison. Though called an acid, it does not redden litmus paper. The crude carbolic acid is commonly used as a disinfectant. This crude acid is a mixture of other coal-tar products with from fifteen to sixty per cent. of carbolic acid. It is a dark-coloured liquid possessing the mixed odour of carbolic acid, and of the coal-tar impurities present. If strong carbolic acid come in contact with the skin, it produces a white appearance, and the surface of the skin is destroyed and peels off, leaving a wound which acquires a brownish colour. If carbolic acid be swallowed in poisonous doses, it exerts both a local and remote action; locally it acts as a corrosive poison; remotely it paralyses the nervous system, death being due to respiratory and cardiac paralysis.

A parliamentary return of the number of deaths in England and Wales caused by the taking of carbolic acid shows, that in the five years from 1887 to 1891 there were 375 deaths from this poison. These were—accident, 138; suicide, 236; murder or manslaughter, 1. Of the cases of suicide, the greater number occurred among females, and attempts at suicide with carbolic acid are also much more frequent among women than among men.

Symptoms.—If strong carbolic acid be swallowed, symptoms come on as a rule at once. (i) Burning pain from the mouth to the stomach; the mucous membrane of the mouth and lips is whitened, and stains are produced on the skin around the mouth, where the acid has come in contact with it. (ii) Vomiting occasionally occurs, but, from the anæsthesia of the gastro-intestinal tract produced by the action of carbolic acid, it is absent in a large majority of the cases. (iii) Respiration is laboured and ultimately stertorous, and there is the peculiar odour of carbolic acid in the breath. (iv) The pupils are contracted, and the conjunctivæ are insensible. (v) Giddiness, collapse, cold clammy skin, and a feeble irregular pulse. (vi) Coma usually precedes death; convulsions sometimes occur. (vii) The urine is usually diminished in amount or suppressed; it is frequently of an olive-green or brownish colour, due to the

conversion of some of the carbolic acid into hydroquinone, and the oxidation of the latter to a brownish product.

Bronchitis and pneumonia occasionally occur as sequelæ or complications, probably from the action of the carbolic acid after absorption. Poisoning may occur from the external application of carbolic acid. Warren[1] mentions a case in which, after the application of carbolic acid to the back of an adult, death occurred in twenty minutes. In another case that came under the notice of the author, a girl, convalescent from scarlet fever, was placed in a bath to which carbolic acid had been added without mixing it with the water; death occurred from the shock produced by the strong carbolic acid coming in contact with the skin. Poisoning has also occurred from inhalation of the vapour. Unthank[2] records a case of a man who was exposed to the vapour of strong carbolic acid for three hours. Giddiness, stupor, convulsions and coma supervened; recovery took place. Bülvebash[3] relates the case of an infant, aged three months, who was brought into a room which had been disinfected by spraying about six pints of a five per cent. carbolic acid solution. In about half an hour the child became exceedingly pale, diarrhœa occurred, and urine of an olive-green colour was passed. Under suitable treatment the infant recovered.

Treatment and antidotes.—The stomach should be emptied by means of a soft stomach-tube, and should then be washed out with either saccharated lime-water or solution of sodium sulphate. If a soft stomach-tube be not available, the ordinary œsophagus-tube of the stomach-pump may be passed, but extreme care should be used, on account of the corroded condition of the walls of the œsophagus, and the consequent danger of rupturing them. External warmth and stimulants should be employed to counteract the collapse; artificial respiration is to be resorted to, if necessary, and milk and other demulcent drinks should be freely given.

[1] *Med. Press and Circ.*, 1882. [2] *Brit. Med. Jour.*, 1872.
[3] *Meditzinskoië Obozrenië*, 1892.

Post-mortem appearances.—Careful search should be made for the stains produced by the poison on the skin around the mouth. The mucous membrane of the mouth and œsophagus is usually softened, white and corroded in parts. The mucous membrane of the stomach is generally hardened, wrinkled, and of a white or brownish colour; in some cases it is found softened and corroded. Small hæmorrhagic points may be found in the mucous membrane, and the contents of the stomach may be blood-stained. The duodenum may present somewhat similar appearances. The brain is occasionally congested, but generally normal in appearance, the fluid in the ventricles mostly possessing the odour of the acid. The lungs are usually congested, and the blood dark-coloured and fluid.

Fatal dose.—One drachm of carbolic acid has caused death. A child six months old has been killed by the administration of a quarter of a teaspoonful of a solution of the acid in glycerine, containing one part of acid to five of glycerine.[1] Recovery has several times taken place after over an ounce of the liquefied acid (90 per cent. strength) has been taken. Davidson [2] relates the recovery of a woman, aged forty, after swallowing four ounces of crude carbolic acid; and Hind [3] relates the case of a girl, aged seventeen, who recovered after swallowing six ounces of crude carbolic acid (14 per cent. strength). Both of these cases received prompt attention.

Fatal period.—Death has occurred in three and in ten minutes. On the other hand, it has been delayed for sixty hours. Usually it occurs in from three to four hours.

Analysis and tests.—Carbolic acid may be separated from organic admixture by distillation with dilute sulphuric acid; from urine it may be removed by agitation with ether. The following tests should be applied to an aqueous solution of the acid:—

I. The odour, which is characteristic.

[1] *Brit. Med. Jour.*, 1882. [2] *Med. Times and Gaz.*, 1875.
[3] *The Lancet*, 1884.

II. The addition of bromine water produces a white precipitate of tribromo-carbolic acid.

III. Ferric chloride gives a violet or purple colour.

IV. On the addition of solution of ammonia and chlorinated lime, and after gently warming the mixture, a bluish tint is produced which turns red on acidulation with an acid.

Picric Acid

This substance is trinitro-carbolic acid, and is obtained by dropping carbolic acid into fuming nitric acid. It is a yellow dye, and is known also as *carbazotic acid*; it is an intensely bitter substance. A few cases of poisoning by it have been recorded, but none were attended with fatal results. Large doses have caused pain in the stomach, vomiting, diarrhœa, intense yellow staining of the skin and conjunctivæ, and red-coloured urine.

Treatment.—The stomach should be emptied and washed out by means of the stomach-pump or stomach-tube. A castor oil purge should be given to remove the picric acid that has passed into the intestines. External warmth and stimulants should be employed to overcome the collapse, and hypodermic injections of morphine should be used, if necessary, to relieve the pain.

Analysis and tests.—Picric acid can be extracted by means of ether from a solution acidified with sulphuric acid. On evaporation of the ether, picric acid is left, and, when dissolved in water, the following tests may be applied:—

I. On gently warming a solution of picric acid with a small quantity of potassium cyanide, the yellow colour changes to a deep blood-red.

II. Solution of picric acid gives with a mixed solution of copper sulphate and ammonia a green precipitate.

Resorcin

In poisonous doses this substance produces giddiness, insensibility, weak pulse, lowered temperature, cyanosis, and a

cold clammy surface. Convulsions sometimes occur. The urine acquires an olive-green colour.

Pyrogallic Acid

This substance as a poison acts by destroying the red blood corpuscles, setting free the hæmogloblin in the liquor sanguinis, and producing hæmoglobinuria. Fatal results have ensued from the use of pyrogallic acid in the form of an ointment. The symptoms, which generally come on in from one to fifteen days after the first application of the ointment, are—nausea, vomiting, diarrhœa, prostration, hæmoglobinuria, anæmia, and great dyspnœa. The latter is probably due to the formation of thrombi.

Salicylic Acid

The sodium salt of this acid is very commonly used in medicine. In large doses it occasionally produces poisonous effects. The symptoms may consist of hæmorrhages of various kinds, such as epistaxis, hæmaturia, hæmorrhages into the retina, mental disturbances, and insensibility. Nausea, vomiting, noises in the ears, deafness, and collapse have resulted from the administration of large doses of sodium salicylate. It is believed by some observers that the poisonous effects are due to the use of artificially prepared salicylic acid, and that they do not accompany the employment of the natural acid.

Salol

This substance is salicylic phenol, and, although generally considered a fairly safe antiseptic and antipyretic, it may produce poisonous effects. Aufrecht and Behm have reported a case in which death followed its use in acute endocarditis. Chlapowski has also recorded a case in which a fatal result followed a fifteen-grain dose of salol. The symptoms consisted of vomiting, dilatation of the pupils, irregular pulse, unconsciousness, and dark urine. Death occurred twelve days later.

CHAPTER XXII

Poisoning by creasote—Acetic acid—Tartaric acid—Amyl alcohol—Amyl nitrite—Amyl acetate—Paraldehyde—Chloralose—Pyridine.

Creasote

Creasote is the name applied to a mixture of substances obtained from wood-tar, of which the chief are guaiacol and cresol. In poisonous doses it causes vomiting, epigastric pain, and diarrhœa. A few deaths from creasote poisoning have been recorded. If a person has lived several hours after swallowing the poison, its peculiar odour may have entirely disappeared from the viscera when they are examined after death.

Acetic Acid

In the concentrated form, as glacial acetic acid, this substance is a corrosive and irritant poison. A few cases of poisoning by it have been recorded. Orfila has recorded a case in which concentrated acetic acid was taken by a female, aged nineteen. She complained of pain in the stomach, suffered from convulsions, and died in a short time. At the post-mortem examination the mucous membrane of the stomach near the pylorus was almost black, and the vessels were filled with dark coagulated blood. In 1893 a case of acetic acid poisoning, which was not attended with a fatal result, occurred in Northampton. Particulars of the case were supplied to the author by Mr. Henry Cropley. A young woman, aged twenty-one, took about two teaspoonfuls of glacial acetic acid, and a few minutes afterwards was seen by Mr. Cropley. She then com-

plained of pain in the throat and epigastric region, and there was a strong odour of acetic acid coming from the mouth, but the mucous membrane of the lips and tongue was not whitened. An antidote of magnesia stirred up in water was immediately administered, and shortly afterwards she vomited, the vomit smelling of acetic acid. Laboured inspiration soon came on, and half an hour later she was coughing up large quantities of mucus, and had great difficulty in swallowing. The back of the throat, soft palate, tonsils, and uvula were red and swollen. During the next forty-eight hours large quantities of muco-purulent matter were coughed up, and later she developed a typical patch of pneumonia at the base of the left lung, which, however, cleared up in about a week. On the third day after taking the acid there was some ulceration on the left side of the uvula, on the left tonsil, and on the left side of the pharynx. For several days she complained of pain on swallowing. In all probability the acid, which was taken from a bottle in one gulp, must have been thrown to the back of the throat, the front part of the mouth escaping, and either some of the vapour of it was drawn into the lungs, or a small quantity passed down into the air passages. In 1893 the author investigated, on behalf of the Treasury, a fatal case of poisoning with glacial acetic acid, in connection with which a servant girl named Newber was tried at the Central Criminal Court for the murder of a child aged ten months (*Reg.* v. *Newber*, C.C.C., 1894). The accused was convicted of the manslaughter of the child with acetic acid. About a teaspoonful of the glacial acid was poured into its mouth, and shortly afterwards the child was found with extensive whitening and softening of the mucous membrane of the tongue, palate, and pharynx, and it also had great difficulty in breathing. Vomiting occurred, the vomited matter containing acetic acid. Owing to the difficulty of breathing, tracheotomy had to be performed, but the child subsequently succumbed. At the post-mortem examination the mucous membranes of the mouth, epiglottis, and larynx were found eroded in patches. Erosions were also found in

the mucous membrane of the œsophagus and stomach. The mucous membrane of the stomach was also corrugated in places, and showed patches of congestion. In 1894 a boy, aged four and a half years, died from drinking an unknown quantity of glacial acetic acid. Death occurred the following day.

Tartaric Acid

This substance is not generally regarded as a poison, but, when taken in large doses and in a concentrated solution, it is capable of acting as an irritant, and of destroying life. Taylor and Stevenson relate a case of fatal tartaric acid poisoning which was the subject of a trial for manslaughter (*Reg.* v. *Watkins*, C.C.C., 1845). The accused gave to the deceased, a man aged twenty-four, one ounce of tartaric acid dissolved in half a pint of warm water; a burning pain was felt in the throat and stomach immediately after swallowing the acid. Soda and magnesia were administered, but vomiting set in and continued until death, which took place nine days afterwards. At the post-mortem examination nearly the whole of the alimentary canal was found highly inflamed. Trevithick[1] reports a fatal case of poisoning by tartaric acid from ignorant self-administration by a woman, aged sixty-seven. The woman owned to having taken about two teaspoonfuls, which would be between 140 and 180 grains, but it was not improbable that a larger dose had in reality been swallowed. It was taken in very strong solution. The symptoms, at the outset, were acute abdominal pains, vomiting, and diarrhœa. Twenty-four hours later she was suffering from intense abdominal pain, and the temperature was 100° F. On the fourth day the patient became delirious, and remained so until her death. On the same day the pulse began markedly to fail. The diarrhœa, which had been checked by opium and bismuth, recommenced on the day before death. At the post-mortem examination acute inflammation of the whole of the alimentary canal was found, the

[1] *Brit. Med. Jour.*, 1893.

mucous membrane of the small and large intestines being especially affected. There were a few shallow erosions of the œsophagus, and in the stomach there were several patches of subserous hæmorrhages. The transverse and descending colon appeared to have suffered more severely than any other part of the intestines.

Amyl Alcohol

This body (which derives its name from *amylum*, starch) is the fusel oil obtained in the preparation of ordinary alcohol from the different forms of starch. It is contained in crude or raw whisky, but the sugar obtained from potato starch yields a more considerable amount, and hence this alcohol is sometimes called *potato oil* or *potato spirit*; it is also known as *oil of grain* and *faints*. It is an oily colourless liquid, possessing a peculiar and unpleasant odour. It is not appreciably soluble in water, but floats on the surface of it like an oil, hence its name *fusel oil*. The vapour is very irritating to the respiratory passages and lungs, and produces, after inhalation, cough, headache, and giddiness. Ord [1] records a case of poisoning by fusel oil, in which recovery took place. The man, sixty-four years of age, drank about half a pint of a mixture of equal parts of fusel oil and spirit. He felt no ill effects for four hours and a half, when he became unconscious. When seen two hours and a half later he was quite unconscious; the muscles of the arms and legs were somewhat rigidly fixed in an extended position; there was no arching of the back, the teeth were tightly clenched, the face bluish, the breathing shallow and infrequent, the pulse could only just be detected at the wrist. The pupils were small, the surface of the body was cold, his breath had a peculiar odour something like that of nitrite of amyl, or of pear drops. The stomach was washed out, a quantity of clear liquid being removed, which had a strong smell. Half an hour later the breathing ceased, and artificial respiration was resorted to. During the next hour stoppage of respiration occurred several

[1] *The Lancet*, 1889.

times, and artificial respiration was kept up during the greater part of the time. Twenty minims of ether were injected hypodermically, and an ounce of brandy given by the rectum. The patient remained unconscious for eleven hours, at the end of which time the breath still smelt strongly of some spirit, and of nitrite of amyl, or the essence of the Jargonelle pear. The urine drawn off by a catheter was high coloured and had the same peculiar odour as the breath. During the night the patient slept well and sweated profusely, the sweat having the same odour as the breath. There was no diarrhœa.

Swain [1] records a fatal case of poisoning by 'faints.' Faints is the latter portion of the distillate obtained on rectifying crude spirits of wine from fermented potatoes, &c. It consists chiefly of fusel oil mixed with large quantities of the secondary and primary propyl alcohols and some of the higher alcohols. The deceased, a man aged fifty-seven, had been a heavy drinker, and was, and had been, in the habit of drinking heavily of faints, which could be obtained in large quantities from a distillery for nothing. At the post-mortem examination a strong smell resembling nitrite of amyl, which pervaded all the organs of the body, was noticed. The kidneys were rather dark; there was no diminution in the cortex, and the capsule peeled off easily. The liver was normal, and weighed sixty ounces. The mucous membrane of the stomach was soft and thick, and the contents consisted of about three ounces of a reddish grumous material containing blood. The ventricles of the brain contained about one ounce and a half of clear fluid possessing the odour above mentioned.

Amyl Nitrite

Rosen [2] records a case of a young man, aged twenty-two, who swallowed about four fluid drachms of nitrite of amyl while suffering from an epileptic seizure. Vomiting occurred; when seen, the face was pale, the lips bloodless, and the pulse 110 a

[1] *Brit. Med. Jour.*, 1891. [2] *Centralbl. f. Klin. Med.*, 1888.

minute. There was pain in the head, a burning sensation in the throat, and pain in the epigastric region. Where the poison had come in contact with the mucous membrane there was slight erosion. The patient suffered from gastric catarrh, but eventually recovered. Shoemaker[1] reports the case of a man, aged sixty, who by mistake swallowed a teaspoonful of amyl nitrite. When seen three minutes later the face was moderately flushed, the pulse was 112, and he complained of a little headache. Copious vomiting was induced, digitalis and brandy were injected hypodermically, and hot bottles were applied to the feet. Sulphate of strychnine in doses of $\frac{1}{30}$ of a grain was given frequently. The man seemed drowsy and stupid, but consciousness was never lost; recovery took place.

Amyl Acetate

This substance is used in confectionery under the name of *essence of Jargonelle pear*. Taylor records the case of a child who on two occasions, after eating some confectionery, which it was calculated contained about one drop of this essence, became partially comatose, with livid lips and a feeble pulse.

Paraldehyde

Paraldehyde is a colourless fluid soluble in water; it acts as an hypnotic similar to chloral. Rolleston[2] records a case in which, an hour after the administration of one fluid drachm of paraldehyde to a patient suffering from chronic bronchitis and emphysema, there occurred sudden dyspnœa and collapse. Mackenkie[3] relates a case of a lady who by mistake took three ounces and a half of paraldehyde. Nine hours later the breath had a very strong smell of paraldehyde; the face was slightly flushed, the pupils were moderately contracted and insensible to light, the pulse was 120, the respiration 40, and the skin

[1] *Philadelphia Med. News*, 1893.
[2] *Practitioner*, 1888. [3] *Brit. Med. Jour.*, 1891.

warm. She was wholly unconscious, and could not be aroused. Strychnine was injected hypodermically, ammonia applied to the nostrils, and faradic electricity resorted to. The urine on being drawn off was found to be redolent of paraldehyde. Thirty-four hours after taking the dose slight consciousness returned, but it was not until an interval of forty-one hours had elapsed that she regained sufficient consciousness to speak; recovery ultimately took place.

Chloralose

Lang[1] reports a case in which a lady suffered from symptoms of poisoning after taking ten grains of chloralose. She had previously taken as much without ill effects. On the occasion referred to she had taken ten grains in two doses with an hour's interval between the two. When seen three hours and a half later she was in a semi-comatose condition, and the face was congested and bluish, the pupils were equal and somewhat dilated, pulse 60, and the skin was warm and moist. After the stomach had been washed out, and an enema of hot coffee administered, recovery soon occurred. Touvenaint[2] reports a case of a diabetic patient, aged sixty, who, after taking three grains of chloralose, suffered from general tremor, incoherent speech, inability to move, nausea, dysphagia, deafness, coldness of the surface, and involuntary passage of urine and fæces. Recovery took place after the employment of enemata of black coffee, and injections of caffeine and ether.

Pyridine

Helme[3] records a fatal case of poisoning by this substance. Commercial pyridine is a colourless volatile fluid, with characteristic odour and taste, and is sometimes used to render alcohol undrinkable. The subject of the case was a man,

[1] *Brit. Med. Jour.*, 1893. [2] *Jour. des Practiciens*, 1894.
[3] *Brit. Med. Jour.*, 1893.

aged twenty-nine, who was employed as a stillman at some tar works, and who accidentally swallowed half a cupful of pyridine. He subsequently vomited several times, and when seen seven hours and a half later he was pale, with slightly cyanosed lips, a dry white tongue, and was perspiring freely. The temperature was 103·4°, the pulse was 128, and was weak and intermittent, and the respirations were 40. The breathing was noisy, due to coarse mucous râles. He complained of tightness in breathing, a choking sensation, pain down the centre of the chest, and pain over the stomach. Demulcents were given and a brandy enema administered. White and frothy expectoration, possessing the odour of pyridine, was coughed up, and the breath had a similar odour. The following day his temperature was 104°, but gradually subsided during the day; the expectoration became purulent, and the lungs showed signs of acute congestion and bronchitis. A few hours later the temperature rose to 105·8°; the patient became delirious, and died forty-three hours after the accident. At the post-mortem examination, the larynx, trachea and bronchi were found lined with a friable yellow membrane, and the bronchi contained purulent matter. The lungs were congested and œdematous. The œsophagus and cardiac end of the stomach were greatly congested, and the pyloric end and commencement of the duodenum slightly so.

GASEOUS POISONS

CHAPTER XXIII

Poisoning by carbon dioxide—Carbon monoxide—Water gas—Sulphuretted hydrogen—Coal gas—Nitrous oxide—Gases of explosives.

In cases of death from the gaseous poisons, a fatal result is generally stated as being due to suffocation. In reality, poisonous gases cause real poisoning, the toxic substance being taken into the lungs and then absorbed into the circulation, instead of being taken into the stomach and thence absorbed, as with the majority of other poisons.

Carbon Dioxide or Carbonic Acid Gas

This gas is a product of combustion and respiration; it is a large constituent of ground air, and is the principal constituent of *choke-damp* resulting from gas explosions in coal-mines. It is also a product of various chemical processes, and may escape into the air during the manufacture of aerated waters, and during the process of fermentation. With carbon monoxide it is evolved from lime-kilns, brick-kilns and cement-kilns. Carbon dioxide is a colourless gas, possessing a peculiar faint pungent odour in quantity, and a faint acid taste. It is heavier than air. The amount of carbon dioxide normally present in the air is very small, being four parts in 10,000 or ·04 per cent. An amount slightly in excess of this in the air of inhabited rooms must be regarded as an impurity, not so much on account of the carbon dioxide itself, as because it is derived from the respiration of human beings; for in the air exhaled from

the lungs putrefiable organic matters are present, and these organic substances have a decidedly deleterious effect on the health of human beings breathing such air. When carbon dioxide in the air of an inhabited room exceeds ·06 per cent., these putrefiable organic matters may become noticeable by a faint smell, which renders the air stuffy or foul. Since these organic matters are extremely difficult to estimate, the determination of the amount of carbon dioxide present becomes a useful gauge of the foulness of the air. Carbon dioxide is not a poison if swallowed into the stomach; in the form of effervescent waters and other drinks it is taken in considerable quantities, but if inhaled in quantity, it produces death from suffocation, on account of its diluting down, and taking the place of, the necessary oxygen of the air. In cases where carbon dioxide is contained in large quantities in a confined portion of air—as in the air of a well, or in vats used for brewing purposes—a rough test is to ascertain whether the air is capable of supporting the combustion of a candle. If the candle be extinguished in such an atmosphere, then it would be extremely dangerous for a human being to descend into it; but if the candle continue to burn, it does not by any means follow that the atmosphere can be breathed even for a short time with impunity, since a candle will burn in air containing 10 to 12 per cent. by volume of carbon dioxide, and although such a mixture might not prove immediately fatal to a human being, yet it would soon cause insensibility and ultimately death to anyone breathing it.

Symptoms.—If pure carbon dioxide be inhaled, it produces spasm of the glottis and rapid death from asphyxia, unless the inhalation of the gas is immediately stopped. If present in poisonous amounts in the air, the following symptoms are produced:—(i) A sensation of pressure within the head, accompanied by noises in the ears. (ii) Vertigo, loss of muscular power, stupor and coma. (iii) The respiration is laboured, and the face is livid or occasionally pale.

Taylor and Stevenson mention the case of a boy who fell

into a large brewing vat among some wet hops, and speedily died from respiring the atmosphere of carbon dioxide; two men successively endeavoured to rescue the boy, but each died in the attempt. A case is also mentioned by Taylor and Stevenson of a man who descended into a large vat, having previously applied the candle test; he was heard to cry out 'There is gas here,' and instantly fell back dead. Biggam[1] relates the occurrence of two deaths from carbon dioxide discharged from the ground into a house. On arriving at the house he found a girl, aged nineteen, already dead, and the other occupants of the house were suffering from the following symptoms:—Intense headache, giddiness, nausea, vomiting, and pain in the region of the stomach. One of these persons died on the fourth day without having recovered consciousness. These accidents indicate the importance of protecting houses from ground emanations, especially in mining districts where these cases occurred. The occurrence took place after several weeks of continuous frost, when the ground was rendered more or less impervious except under the houses.

Treatment.—This consists in immediate removal from the poisonous atmosphere; artificial respiration should be carried on at once, with inhalations of oxygen if possible; stimulants should also be administered.

Post-mortem appearances.—Those of death from asphyxia (see ii. p. 106). The venous system and right heart are found filled with dark-coloured liquid blood; the lungs are congested.

Carbon Monoxide or Carbonic Oxide.

Carbonic oxide is the poisonous constituent of a great number of mixed gases. The burning of charcoal produces carbonic oxide mixed with carbon dioxide. It is also produced by burning coke, and is contained in the gases evolved from blast-furnaces, and from regenerator-furnaces. It is also contained in gases evolved from slow-combustion stoves and

[1] *Brit. Med. Jour.*, 1893.

gasoline stoves. Coal gas contains a certain amount of carbonic oxide, to which its poisonous properties are mainly due. Water gas, obtained by passing steam over heated coke, contains about 40 per cent. by volume of carbonic oxide, the remainder being principally hydrogen. Carbonic oxide is a colourless, odourless, and tasteless gas; it is extremely poisonous, and, on account of its possessing no odour, the deadly atmosphere may be inhaled without the person inhaling it being aware of the presence of this gas. Its effects are insidious, because, in addition to its being destitute of odour, it exercises no irritating effect on the air-passages, but rather acts as a narcotic, so that it may be breathed without any alarm being excited in the victim. One of its effects is to cause loss of the power of movement, and even of desire to make any exertion. Even if present in the atmosphere in a very small quantity, it may produce a fatal result, if that atmosphere be respired for a long period; this is due to the carbonic oxide possessing the power of uniting with the hæmoglobin of the blood, forming a fixed compound, which is incapable, except after a prolonged period, of taking up oxygen from the air. In the case of a person breathing an atmosphere containing only a small amount of carbonic oxide, the hæmoglobin of the blood would select the carbonic oxide from the air drawn into the lungs, and so in time the blood would become charged with this compound of carbonic oxide hæmoglobin. One-tenth per cent. of carbonic oxide in an atmosphere is dangerous, and, according to Stevenson, is capable of destroying life. A somewhat common method of committing suicide in France and some other countries consists in inhaling the fumes of burning charcoal, placed in the centre of a closed and non-ventilated room. In this way the atmosphere of the room becomes impregnated with a mixture of carbon monoxide and carbon dioxide, the former, however, being the principal poisonous constituent. An analysis of the atmosphere of such a room has yielded 4·6 per cent. of carbon dioxide, and ·5 per cent. of carbon monoxide. Water gas, as previously stated, is a mixture mainly of carbonic

oxide and hydrogen, containing about 40 per cent. by volume of the former gas. Water gas is especially a dangerous gas to employ in houses, since, on account of the absence of odour, leakages of it are not detected except by the production of poisonous symptoms or fatal results. In the United States many fatal accidents have occurred from its escape into the air of rooms.

Symptoms.—On account of the peculiar narcotic influence of carbonic oxide when absorbed into the blood, which causes a loss of power of movement, and even of the desire to make any exertion, a person exposed to its influence may be powerless to escape from the poisonous atmosphere. The following are the symptoms generally noticed:—(i) Headache, giddiness, noises in the ears, nausea, and occasionally vomiting. (ii) Great prostration, insensibility and coma. In fatal cases convulsions may precede death.

Ruata[1] reports a case in which a patient for five days showed signs of active delirium without any apparent cause. It was afterwards ascertained that he had for some days previously been exposed to the fumes of a coke stove in a closed room, and that during this time he complained of frontal headache and became very depressed.

Treatment.—The patient should be removed from the poisonous atmosphere at once; artificial respiration should be resorted to, and oxygen inhalations given from time to time. Stimulants should be administered. Two cases have been recorded in which subcutaneous injections of nitroglycerine were followed by recovery, and another case has been recorded in which transfusion was followed with fairly satisfactory results.

Post-mortem appearances.—The post-mortem stains have a peculiar bright pink colour. Apart from the colour of these stains, the skin is pale; the eyes are bright and staring, the pupils dilated, and the jaws fixed. The blood is of a bright red or cherry-red colour, the colour being due to the compound of

[1] *Gazz. Med. di Torino*, 1892.

carbonic oxide with hæmoglobin. The viscera are also of a bright red colour on account of their being charged with the coloured blood. Rosy blood-stained fluid may be found in the stomach, bladder, and cranial cavity. Rigor mortis is usually prolonged and putrefaction is delayed. Stevenson[1] states that portions of the liver from a case of water-gas poisoning showed an unchanged aspect, and retained the odour of the fresh liver seven weeks after death, although no preservative had been used; portions of the stomach and duodenum were at that time unchanged in appearance; he describes the keeping powers of the viscera as phenomenal. The bright colour of the blood remains for a long time, and the rosy hue of portions of the viscera has been observed by Stevenson after a lapse of seventeen months.

Spectroscopic examination of the blood.—Blood which is fully saturated with carbonic oxide shows, when examined by the spectroscope, two bands of carbonic oxide hæmoglobin, which somewhat resemble those of oxyhæmoglobin, although they are rather nearer the violet end of the spectrum, and are more refrangible than the latter (see Frontispiece). In examining blood for carbonic oxide, it is best to use a spectroscope which enables the two spectra to be seen superposed—that of the blood under examination, and that of normal blood. The differences in position and refrangibility of the bands of carbonic oxide hæmoglobin and oxyhæmoglobin are then clearly seen. On the addition of a reducing agent, such as Stokes's solution, or yellow ammonium sulphide, the spectrum of the carbonic oxide hæmoglobin remains unchanged, whereas that of the oxyhæmoglobin changes to a one-banded spectrum. It should be borne in mind that the spectroscopic test works with absolute accuracy with blood fully saturated with carbonic oxide, but if the blood be not fully saturated with that gas, then there is present a mixture of carbonic oxide hæmoglobin and oxyhæmoglobin, and, on the addition of a reducing agent, the hæmoglobin in union with the oxygen becomes reduced, so

[1] *Guy's Hosp. Repts.*, 1890.

that in such a case the spectrum undergoes some change on the addition of the reducing agent, the reduced hæmoglobin showing the broad band upon which are superimposed the two bands of the carbonic oxide hæmoglobin (see Frontispiece).

Another test for blood containing carbon monoxide is to dilute one part of the blood with nineteen parts of distilled water, and then add an equal volume of sodium hydrate solution (sp. gr. 1·34); the solution containing carbon monoxide blood, after a momentary turbidity, becomes bright, and light red in colour; that containing ordinary blood changes to dirty brown.

Sulphuretted Hydrogen

In the pure state this gas is quickly fatal. Sewer gas contains sulphuretted hydrogen, to which its toxic effects are chiefly due.

Symptoms.—If inhaled in the diluted state, sulphuretted hydrogen causes nausea, sickness, pain in the head, difficulty in breathing, giddiness, and insensibility. The secondary effects are rare. Wiglesworth[1] records the case of a man employed in some chemical works, who, after having inhaled sulphuretted hydrogen by accident, became maniacal, and continued so for two or three weeks; he did not recover his full mental vigour until about five months had elapsed. In cases of poisoning by this gas death is probably due to paralysis of the central nervous system, affecting especially the lungs and heart.

Treatment.—Removal of the patient from the poisonous air. Artificial respiration and inhalations of oxygen should be employed; stimulants should also be administered.

Post-mortem appearances.—The blood is liquid and of a brownish-black colour, from conversion of the iron of the hæmoglobin into sulphide of iron. Putrefactive changes quickly follow death.

[1] *Brit. Med. Jour.*, 1892.

Coal Gas

Coal gas consists of about 90 per cent. by volume of diluent gases, and about 6 per cent. by volume of illuminants, which consist of hydrocarbon gases or vapours rich in carbon, to the presence of which the luminosity of coal gas, when burnt, is due. The 90 per cent. of diluent gases is made up of 50 per cent. of hydrogen, 35 per cent. of marsh gas, and 5 per cent. of carbonic oxide, and it is to the latter gas that the poisonous properties of coal gas are mainly due. The majority of cases of poisoning by coal gas occur from the escape of gas into the atmosphere of rooms from the tap not having been turned off, or from its having been turned on without lighting the gas. Templeman[1] relates a case of poisoning by coal gas in ground air, in which the gas escaped from a broken pipe which was thirty inches below the surface and three feet from the wall of the house. There had been a prolonged and severe frost, so that the ground was very hard, and this prevented the gas escaping into the outside air; but as no provision had been made to prevent the entrance of ground air into the house, the mixture of coal gas and ground air had been drawn in. The house possessed only one room, the occupants of it consisting of two females and a man, all of whom slept for some hours in the room which was impregnated with the coal gas. When seen by Templeman they were all unconscious, the extremities were cold, their pulses imperceptible, and the pupils were extremely contracted. The man died about ten hours, and one of the females twenty-seven hours, later.

Symptoms.—The symptoms produced by the inhalation of a mixture of air and coal gas are—(i) Giddiness, headache, nausea, and vomiting. (ii) Contracted or dilated pupils, clenched jaw, froth issuing from the mouth, and odour of the gas in the breath. (iii) Collapse, confusion of intellect, loss of consciousness, and convulsions.

As with carbonic oxide poisoning, the breathing of coal gas

[1] *Brit. Med. Jour.*, 1893.

produces a condition of stupefaction, and so renders a person unable to save himself by withdrawal from the poisonous atmosphere.

Broadbent[1] reports the case of a man, aged forty-three, who had slept all night in a small unventilated room with gas turned on. When seen he was unconscious and could not be roused, the face was flushed, lips red, pupils not dilated and reacting to light. Some hours later his consciousness had not returned, and oxygen inhalations were given ; twelve ounces of blood were removed by venesection, and the following day twelve ounces of defibrinated human blood were transfused. After this, consciousness slightly returned, and he was able to speak a little. Two days after the poisoning with the gas he was able, after much rousing and stimulation, to speak a little, but when left alone relapsed instantly into a state of stupor. For ten days his condition improved, but he then relapsed ; paralysis, especially of the arms and legs, came on, accompanied by extreme muscular wasting. On the eighteenth day after the poisoning by the gas he passed into a condition of coma, and died on the nineteenth day. At the post-mortem examination softening of the lenticular nucleus and of the posterior part of the internal capsule was seen on naked-eye inspection.

Treatment.—The patient should be immediately removed from the poisoned atmosphere; the tongue should be pulled forward, and artificial respiration resorted to. External warmth and stimulants should be employed. Galvanism may also be resorted to, one pole being applied to the nape of the neck and the other over the epigastrium. In cases of poisoning by coal gas the blood is found liquid, and of a bright red or cherry-red colour, due to the formation of carbonic oxide hæmoglobin. Usually there is congestion of the brain and its meninges, the surface of the brain being generally of a bright red colour. The mucous membrane of the respiratory tract is generally strongly injected, and the substance of the lungs is of a bright red colour.

[1] *Brit. Med. Jour.*, 1893.

Nitrous Oxide, or Laughing Gas

This gas is commonly employed as an anæsthetic in minor operations, and especially in connection with the extraction of teeth. As an anæsthetic laughing gas operates by producing temporary asphyxia—that is, it prevents oxidative changes in the blood, and, as in death from asphyxia, respiration is arrested before the heart ceases to beat. On very rare occasions the administration of nitrous oxide has produced fatal effects ; in the majority of such cases some previous weakness or disease of the heart is responsible for the fatal result.

Gases of Explosives

With the majority of explosives, the bulk of the gases generated consists of carbon dioxide and nitrogen. The gases generated by the explosion of gunpowder consist of carbon dioxide, nitrogen, carbon monoxide, potassium sulphide (forming the smoke), and some cyanogen compounds. From the potassium sulphide a little sulphuretted hydrogen is liberated, imparting the peculiar smell to the gunpowder smoke.

Gun-cotton, nitroglycerine, and dynamite, when exploded, yield a mixture of carbon dioxide, carbon monoxide, and nitrogen. Roburite and tonite (see p. 361) are compounds that are frequently used in coal-mines as substitutes for gunpowder for blasting purposes. The subject of the composition of the gases evolved by the explosion of these compounds has recently been investigated by a committee appointed to ascertain whether their use is likely to have a deleterious effect on the health of the men employed in blasting operations. Bedson, who conducted the chemical part of the examination, found that the vitiation of the air with carbon dioxide after the explosion of roburite or tonite was much the same as in the case of gunpowder, and that traces of carbon monoxide could also be detected. With the ventilation provided in the mines, the fumes were speedily dissipated, so that roburite and tonite do

not present any special dangers as compared with gunpowder, except for the fact that the fumes from roburite are much less visible, and the miners might therefore be tempted to return too soon after the explosion. The committee therefore came to the conclusion that the products from the explosion of roburite, tonite, and conite are not more deleterious than the products of the explosion of gunpowder; that, with regard to the effects of the explosion of roburite, no chemical evidence was obtained of the presence of nitrobenzene, and throughout the enquiry no case of nitrobenzene poisoning was met with. Hatton[1] reports a fatal case of inhalation of roburite fumes. The deceased, a miner, after firing a roburite shot and allowing eight minutes to elapse, proceeded to take down the hanging coal. A few minutes later he complained of pain in the head and faintness. Next day he was suffering from dizziness, pain in the head, and marked cyanosis about the lips; his articulation was thick and almost unintelligible. The pain in the head increased in severity, coma rapidly supervened, and the patient died on the third day. At the post-mortem examination, made on the second day after death, the only noticeable point on external examination was the cyanosis of the lips. The dura mater was inflamed, and there was intense venous congestion on the surface of the brain, with some superficial encephalitis; punctiform ecchymoses were found throughout the brain. On opening the thorax there was no odour perceptible. The lungs were congested, but the remaining organs were healthy. There was no trace of carbon monoxide in the blood, nor of the presence of any nitrobenzene compounds.

[1] *The Lancet*, 1892.

ANIMAL POISONS

CHAPTER XXIV

Poisoning by cantharides—Snake poisons—Ptomaines, or animal alkaloids—Ptomaines and disease—Poisonous and infected foods.

Cantharides

Cantharis Vesicatoria is the *Spanish fly* or *blister-beetle.* When taken in the form of the solid fly powder, or of a preparation such as the tincture or acetum cantharidis, Spanish fly is an irritant to the gastro-intestinal tract and to the urinary organs. It is sometimes employed as an abortive and also to excite sexual feeling. With regard to its use as an abortive, pregnant women have been known to abort after administration of cantharides, but in all probability it possesses no true ecbolic properties. The expulsion of the uterine contents are, in such cases, most probably brought about as the result of the intense irritant effect of the cantharides, especially upon the intestinal tract. In the majority of the cases where abortion has been procured by means of cantharides, the life of the woman has been sacrificed. The active principle of cantharides is cantharidin. Cantharides kills by producing paralysis of the respiratory centres.

Symptoms.—(i) Burning pain in the mouth and throat, with difficulty of swallowing. (ii) Violent pain in the abdomen, nausea, and vomiting of blood-stained mucus. (iii) Great thirst, salivation, and purging, with tenesmus, may occur. (iv) Dull pain in the loins, and a constant desire to micturate, a few

drops of blood-stained urine being passed, accompanied by strangury. (v) Irritation of the genital organs, accompanied by swelling and heat. (vi) Giddiness, faintness, delirium, and convulsions.

Treatment.—The soft stomach-tube should be passed and the stomach emptied and washed out; if the tube be not available, an emetic of mustard and water, or a hypodermic injection of apomorphine, may be administered. Stimulants should be given, and morphine or opium to relieve the pain.

Post-mortem appearances.—Careful search should be made in the contents of the stomach and intestines for shining green particles of the insect. The mucous membrane of the stomach and intestines is generally inflamed, and may show marks of erosion. Inflammation of the kidneys, uterus, bladder and urethra is generally evident.

Fatal dose.—Twenty-four grains of cantharides in powder have proved fatal to a young woman. One fluid ounce of the tincture has caused the death of a youth aged seventeen.

Analysis and test.—Cantharidin may be extracted from organic admixture, such as the contents of the stomach, by Dragendorff's method, which is conducted as follows:—the organic mixture is boiled with caustic potash and water, filtered, acid added to the filtrate to set free the cantharidin from its union with the caustic potash, and the filtrate boiled with four times its volume of alcohol. After cooling, the alcoholic solution is filtered, and the filtrate is evaporated and the residue extracted with chloroform to dissolve out the cantharidin. On evaporation of the chloroform extract the cantharidin is left, and may be detected by its vesicating properties. To do this the residue should be dissolved in a drop or two of almond oil or olive oil, and a drop of the solution taken up with a small quantity of cotton wool; the wool is then applied to the inside of the arm, a piece of oil-silk placed over the wool, and the whole strapped to the arm with some sticking-plaster. After some hours the effect may be examined, when a blister will have been raised if only a very minute amount of cantharidin has been present.

Snake Poisons

Cobra poison.—After a bite from this poisonous snake there is frequently very little sign of external injury. A slight scratch or puncture indicates the site of the bite, and the areolar tissue beneath is of a purple colour and infiltrated with purple blood-stained fluid. Outside the purple areola there is generally a bright scarlet ring of inflammation, and this fades into the normal colour of the surrounding skin. These appearances are due to the irritant properties of the cobra poison. The first symptoms following on the bite are local pain and smarting of the bitten part, after which there follows an interval, the average duration of which is about an hour (it may be as short as a few minutes, or as long as four hours). The symptoms then rapidly develop, and consist of a feeling resembling that of intoxication, followed by loss of power over the legs, loss of power over the speech, over swallowing, and of the movement of the lips. The tongue hangs out of the mouth motionless, saliva runs down the face, and, as pointed out by Wall,[1] the condition closely resembles that of glosso-pharyngeal paralysis. Death occurs usually in from one to two hours after the bite.

Common viper poison.—The common viper, which is still found in some parts of Great Britain, produces, as the result of its bite, dilatation of the pupils, lowering of temperature, convulsions, paralysis, and death. The course is usually very rapid.

Death-adder poison.—Death-adder is the most deadly of the Australian snakes. Recently cases of poisoning by this snake have been successfully treated by employing strychnine as an antidote. The action of the poison, no doubt, is to produce paralysis of the motor and vaso-motor centres. Mueller has injected as an antidote strychnine, employing, in less than six hours, quantities of strychnine that would have been fatal but for the antagonism existing between it and the snake poison. In some instances as much as half a grain to a grain of strychnine has been required to turn the scale in favour

[1] *Proc. Roy. Soc.*, 1881.

of the patient. Hunt[1] records the case of a child, sixteen months old, who was bitten on the finger by a death-adder. A few minutes after the bite the top of the finger was removed, the stump sucked, and then drenched with ammonia. When seen three hours later by Hunt, the child was almost comatose, the body and extremities were cold and clammy, the pupils widely dilated and insensible to light, and the pulse rapid, feeble and irregular. Strychnine was hypodermically injected. External warmth was applied, and a strong faradic current applied to the nape of the neck and along the spine. In two hours' time the child was practically out of danger.

The snake poisons are probably albumoses of an extremely toxic nature. No definite chemical composition can as yet be assigned to them, as they have not been isolated sufficiently pure to allow of an exhaustive analysis.

Treatment.—The best treatment in the case of a bite from a poisonous snake is for the wound to be immediately sucked; the saliva, &c. should be spat out of the mouth after sucking, and the mouth rinsed with water. After sucking the wound, it should be bathed with solution of potassium permanganate, to destroy any of the poison that may be left on the surface. Strychnine should then be injected hypodermically, and the administration of it pushed as far as necessary. Galvanism and artificial respiration should be employed if required.

Ptomaines or Animal Alkaloids

The word ptomaine is derived from πτωμα, a dead body. Ptomaines, also known as cadaveric alkaloids, are alkaloids produced by the decomposition of animal substances. The term *ptomaine* was at first restricted to alkaloids produced by cadaveric decomposition, but it is now also employed to designate alkaloids of animal origin formed during life as a result of chemical changes induced by some agency acting within the organism.

[1] *Austral. Med. Gaz.*, 1891.

The term *leucomaine* has recently been introduced to particularise the animal alkaloids formed during life from those produced by decomposition of dead animal matter; but it would be preferable for the terms *ptomaines* and *leucomaines* to be abandoned, and to class these bases of animal origin in one category as *animal alkaloids.*

It is now well known that the power of manufacturing alkaloids is not restricted to plants, but is shared by animal organisms. In 1822 Gaspard and Stick extracted a venomous principle from corpses. In 1856 Panum detected a very active poison in putrid matter. In 1866 Dupré and Bence Jones found an alkaloidal substance, resembling quinine in some of its properties, in the liver. In 1868 Bergmann and Schmiedeberg obtained from putrid beer a nitrogenous crystalline substance, which they called *sepsine*, and which was subsequently thought to be discovered in septicæmic blood. In 1871 Selmi, examining the dead body of a person supposed to have been poisoned, extracted an alkaloid which he was unable to identify with any known body, and was led to suspect that it had been produced after death; and in 1877 Selmi, by subjecting pure albumen to putrefaction, produced and separated two new alkaloids. Since then Gautier and Brieger have made extensive researches, resulting in the discovery of several animal alkaloids.

Creatinine, discovered in urine by Liebig and Pettenkofer, was the first body of animal origin acknowledged to be an alkaloid. Later on, Liebrich detected the already known vegetable alkaloid betaine in normal urine. In 1880 Pouchet detected carnine in the urine of man; and this was confirmed in 1881 by Gautier, who showed that it possessed the general properties of a ptomaine. In 1882 Bouchard demonstrated that, not only were alkaloids present in appreciable quantities in normal urines, but that they augmented notably in the course of certain maladies—typhoid fever, for instance. These results have been confirmed by Lepine and Aubert, and by the author, who has extracted from the urines of typhoid and scarlet

fever patients ptomaines which are present in greatest amount during the height of the fever, but which diminish as the fever subsides.

Ptomaines of known composition.—The common ancestor of alkaloids, whether animal or vegetable, is albumin, the complex albumin molecule being split up, either by bacterial agency or otherwise, into several less complex molecules, among which are the animal alkaloids. The following is a list of the principal ptomaines that have been extracted from putrefying animal matters, and (with the exception of mydaleine) submitted to ultimate analysis :—

Collodine, $C_8H_{11}N$, from putrefying horseflesh and mackerel.

Parvoline, $C_9H_{13}N$, from putrefying horseflesh and mackerel.

Unnamed base, $C_{10}H_{15}N$, from putrefying fibrin of bullock's blood.

Hydrocollodine, $C_8H_{13}N$, from putrefying horseflesh and mackerel.

Putrescine, $C_4H_{12}N_2$, from human corpses.

Neuridine, $C_5H_{14}N_2$, from human corpses, and from putrefying fish and cheese.

Cadaverine, $C_5H_{16}N_2$, from human corpses.

Neurine, $C_5H_{13}NO$, from human corpses.

Mydaleine, from human corpses.

Muscarine, $C_5H_{13}NO_2$, from putrid fish.

Choline, $C_5H_{15}NO_2$, from human corpses.

Gadinine, $C_7H_{16}NO_2$, from putrid codfish.

Animal alkaloids are also a necessary product of vital physiological processes, and have been extracted from the secretions of living beings, and from fresh animal tissues. The following is a list of the principal animal alkaloids so obtained :—

Creatinine, $C_4H_7N_3O$, from urine.

Pseudoxanthine, $C_4H_5N_5O$, from urine and flesh.

Sarkine, $C_5H_4N_4O$, from urine and flesh.

Xanthine, $C_5H_4N_4O_2$, from urine and flesh.

Crusocreatinine, $C_5H_8N_4O$, from fresh meat.
Xanthocreatinine, $C_5H_{10}N_4O$, from fresh meat.
Guanine, $C_5H_5N_5O$, from flesh and guano.
Carnine, $C_7H_8N_4O_3$, from fresh meat.
Betaine, $C_5H_{11}NO_2$, from urine.
Mytilotoxine, $C_6H_{15}NO_2$, from poisonous mussels.

Ptomaines and disease.—Animal alkaloids are being incessantly produced within our bodies as a result of the normal physiological processes, and they are eliminated by the bowels, kidneys, liver, skin, and lungs; but if from any cause these eliminating organs fail to perfectly fulfil their excretory functions, then an accumulation of those alkaloids in the system occurs, and a toxic action is exerted by them on the nervous centres. In this way can be explained the headache resulting from constipation, and the more serious nervous symptoms resulting from deficient excretory action of the kidneys in certain diseases of those organs. The removal of these animal alkaloids is, however, not only effected by the excretory organs, but, in addition, a powerful agent for their destruction is at work in the oxygen of the blood, which is continually oxidising and destroying them; and it seems probable that this combustion to a large extent occurs in the liver. If, while the excretory organs remain sound, there be excessive production of animal alkaloids, but insufficient elimination and destruction—a condition which is obtained in all forms of over-exertion, as in a prolonged march—then accumulation of material, elaborated in excess and imperfectly eliminated or destroyed, occurs, and an auto-infection, a temporary poisoning of the system, results, the poison affecting the nervous centres and producing the fever of over-exertion—the fever of prostration.

As regards the infectious fevers, it is probable that, after the admission of the specific micro-organisms into the body, and provided they find the conditions suitable, they live and multiply, and, as a result of their vital activity, a powerful alkaloidal or other poison is produced, the toxicity of which is

the cause of the symptoms of the disease. If so, each infectious fever is the result of a fermentative decomposition of albuminous matter within the body, induced by a special micro-organism manufacturing its own peculiar poison for each disease.

In addition to these diseases, it seems probable that, in many non-contagious diseases, abnormal chemical changes may take place, independently of bacterial agency, and may result in the formation of poisons which exert a toxic influence on the organism within which they are produced. The toxic agent of expired air is probably a volatile ptomaine, or mixture of ptomaines.

It has been stated that all ptomaines have an action directly opposite to atropine. This, however, is incorrect, since from putrefying meat infusion a ptomaine has been obtained, which, like atropine, causes dilatation of the pupils; mydaleine (obtained from putrefying corpses) also has a like action on the pupils. Lauder Brunton [1] remarks that 'most of the alkaloids which have been obtained by the decomposition of albumen appear to belong to the muscarine type, although some appear to belong rather to the atropine type.'

Extraction of ptomaines.—Ptomaines can be extracted from organic matters by the general process for alkaloidal extraction (see p. 254).

An occasional defence set up in murder trials, when one or other of the rarer vegetable alkaloids has been used as a poison, is that the alkaloidal poison extracted by the toxicologist may have been a ptomaine produced by putrefactive or other changes in the body of the deceased person. Another point of medico-legal interest attaching to the ptomaines arises from a remote liability of their being confounded with some of the vegetable alkaloids, and so leading to mistakes in medico-legal practice. As a fact, however, only two ptomaines that have been isolated from corpses are active poisons, viz.—*neurine* and *mydaleine*. Neurine does not appear until five or six days after

[1] *Disorders of Digestion*, p. 291.

death, and mydaleine not until seven days after, and then only in minute quantities. In the period after death during which post-mortem examinations are usually made, choline is the only ptomaine present, and this is a very feebly poisonous body. Even if putrefactive changes are well advanced, ptomaines are only present in very small quantities, and if extracted, although they answer to many of the alkaloidal group-tests, yet they do not respond to the special tests and groups of tests given by the vegetable alkaloids. It is true that there is no test by which ptomaines as a class can be distinguished from vegetable alkaloids, but, on the other hand, no ptomaine as yet discovered yields the same chemical reactions, and possesses the same physiological properties, as any of the vegetable alkaloids.

Since neurine and mydaleine are the only actively poisonous ptomaines that are likely to be extracted from corpses by the toxicologist, a knowledge of their physiological effects is of some importance. The following account is taken from Dixon Mann's work on 'Forensic Medicine and Toxicology,' pp. 616, 617:— 'According to Brieger, the symptoms in rabbits produced by the subcutaneous injection of *neurine hydrochlorate* are:—moisture of the nostrils and upper lip, followed by movements as in the act of chewing, with the formation of drops of thick mucus at the angles of the mouth; profuse salivation then occurs, which continues to the end. The breathing is at first accelerated, it then becomes shallow and irregular, with marked symptoms of dyspnœa. The heart-beats, much quickened at first, are then slowed and enfeebled, becoming weaker and weaker until contractions finally cease, and the heart stops in diastole, respiration continuing for a time after the heart ceases to beat. Occasionally, but not constantly, the pupils are contracted after injection of the ptomaine; on the other hand, a concentrated solution of it dropped into the eye almost always produces narrowing of the pupil. Active peristalsis of the bowels comes on early. Clonic spasms, which are not altogether respiratory, and paralysis of the hind, and then of the fore, legs precede death. Five milligrammes produce symptoms of poisoning in, and four

centigrammes constitute a lethal dose for, rabbits, weighing one kilogramme. The symptoms strongly resemble those produced by muscarine, and like them are combated by atropine.

'*Mydaleine* produces the following symptoms in rabbits:— The secretion from the nostrils and the lachrymal glands is increased; the pupils are dilated and reactionless, and the vessels of the ears are injected; the temperature is elevated 1° to 2°C.; the pulse and respiration at first increase in rapidity, and then become slower, the heart ceasing in diastole. Peristalsis is increased, and there is diarrhœa and vomiting; clonic spasms and paralysis, with a tendency to stupor, precede death. Five milligrammes of mydaleine hydrochlorate injected into a kitten caused rapid dilatation of the pupils (which were reactionless), diarrhœa, vomiting, salivation, sweating of the feet, stupor, muscular spasms, paralysis first of the hind and subsequently of the fore legs, slow laboured breathing and death. The heart was found in diastole; the mucous membrane of the bowels was somewhat injected, and they contained some thin fluid secretion.'

Several attempts have been made to find some general test for distinguishing ptomaines from the vegetable alkaloids, but, as previously stated, no reliable tests exist. One method brought forward was based upon the rapid reduction of potassium ferricyanide to the ferrocyanide by ptomaines. The ptomaine is converted into a sulphate, and the solution of it is mixed with a drop of solution of potassium ferricyanide in a watch-glass; on adding a drop of ferric chloride solution to the contents of the watch-glass, the deep blue colour of prussian blue is produced, if reduction of the ferricyanide has taken place. Under similar conditions, however, some of the vegetable alkaloids, such as aconitine, morphine, eserine, and hyoscyamine, exert a rapid reducing action on the ferricyanide, and others, such as emetine, igasurine, nicotine, colchicine, &c., exert a slower reducing action.

Brouardel and Boutmy[1] have suggested, as a distinction

[1] *Mon. Scient.*, 3 Sér. ii. pp. 736, 738.

between ptomaines and vegetable alkaloids, making use of their different action on photographic silver bromide paper. The paper is written on with a glass pen dipped in a solution of the alkaloid, and is then kept from the light for half an hour; it is then washed first with sodium hyposulphite solution, and lastly with water. The ptomaines reduce and blacken the silver compound, but the vegetable alkaloids do not. Neither of these processes, however, can be absolutely relied on for distinguishing between ptomaines and vegetable alkaloids.

Poisonous and Infected Foods

It occasionally happens that, under certain and various conditions, different articles of diet become possessed of poisonous properties; this may happen quite apart from the criminal or accidental introduction of poison. Articles of food may become more or less poisonous from the following causes:—

1. The poisonous results may be due to food conveying a true infection, or to poisons developed in and from the food by bacterial agencies.

2. The poisonous results may be due to toxic metallic salts.

3. The poisonous results may be due to the presence in the food of the germs or spores of certain specific diseases.

4. The food may be infected with certain parasites or their ova, which parasites or ova are capable of undergoing development in man.

5. The flesh of animals may contain some poisonous substance administered as a drug or eaten as food.

These various causes will now be discussed in some detail:

1. The poisonous results may be due to food conveying a true infection, or to poisons developed in and from the food by bacterial agencies.

I. *Meat.*—Meat poisoning has resulted from consumption of the following kinds of meat, viz.: pork, ham, sausages, tongue, beef, brawn, veal pie, pork pie, beef pie, and chicken broth. The symptoms produced by the consumption of

poisonous meat are those of gastro-intestinal disturbance, which is more or less severe, and which is accompanied by disturbance of the nervous system. The first symptoms of illness, as a rule, occur rather suddenly at a varying period after consumption of the poisonous food. The symptoms generally are nausea, vomiting, pain in the abdomen, and diarrhœa, the latter usually being of an offensive character. The nervous symptoms, as a rule, are faintness, muscular weakness, prostration, and occasionally rigors. As a rule, these symptoms are followed by fever, headache, and thirst. If the illness progresses, however, other nervous symptoms may be observed, such as cramps, muscular twitchings, disturbances of vision, dilatation of the pupils, drowsiness, and occasionally coma. The appearances observed in the organs of the body after death are inflammatory, hæmorrhagic, or destructive changes in the stomach and intestines; engorgement of the lung-tissues with blood; and inflammatory or destructive changes in the liver and kidneys. This condition must be regarded as not simply the result of the irritant effects of the poisonous food on the stomach and intestines, but also as being due to a general disease resembling in many of its effects a specific fever. This latter point has been insisted on by Sir George Buchanan, who says 'that the phenomena which were spoken of as food poisoning are claiming on ever-growing evidence to be regarded as true infective diseases, as much so as was scarlet fever or tuberculosis; that they have not been generally admitted into this rank arises, firstly, from the circumstance that some of them have seemed to be wanting in the incubation period; and, secondly, because they are rarely recognised as being transmissible from person to person.' An epidemic occurred in Middlesboro in the early part of 1888, and resulted in 490 deaths in a population of 98,000. It was an epidemic of pleuro-pneumonia, due, at all events in part, to the consumption of American bacon, made by soaking in water, and then only slightly drying, salted pork imported from America. In this bacon there was found a bacillus which was capable of

producing a specific general fever, the special characteristic of which was pleuro-pneumonia. Many cases of meat poisoning have been recorded of late years, of which the following have been selected as typical examples :—

Underhill[1] records some cases of poisoning by tinned beef. The tin was not blown, and half a pound of the contents was divided into eight portions, which were partaken of by eight persons, two adults and six children. In periods varying from two and a half to four hours after eating the beef, all the persons were seized with illness. The symptoms were, profuse vomiting, intense abdominal pain, profuse diarrhœa, coldness of extremities, and collapse. The urgent symptoms lasted about six hours. Underhill found that down one side of the tin the meat was discoloured on the surface, but otherwise it was perfectly red and good, and did not smell. Several cases of fatal poisoning from roast pork, in connection with which nothing unusual was noticed in the meat at the time of eating, have been described. At Whitchurch, in 1878, several people were taken ill after eating from a roast leg of pork, two of them dying shortly afterwards. At Nottingham, in 1881, several people were taken ill under precisely similar conditions. The period elapsing between eating the pork and the first definite symptoms of illness varied from twelve to thirty-four hours, and one of the sufferers died after four days' illness. At Battersea, in 1893, three people were attacked with illness after eating baked pork. They noticed nothing unusual with the meat, but, about twelve to twenty-four hours after eating the pork, they were seized with diarrhœa, and one of them subsequently died.

Bristowe[2] records details of an outbreak of illness at Camberwell after eating pork pies. Thirty-seven cases occurred, two of which were fatal. They were all traced to one batch of pork pies, sold by a respectable tradesman, and received by him from Leicester. The symptoms were, abdominal pain, sick-

[1] *Brit. Med. Jour.*, 1892. [2] *Ibid.*, 1893.

ness, diarrhœa, severe headache, high temperature, and collapse. Although, as a rule, the symptoms did not appear until twenty-four or thirty-six hours after eating the pies, several cases occurred in which the illness came on almost at once, in a comparatively slight form, and was followed by an intermission, the serious attack only coming on, as in the other cases, at a later period. One woman, whose husband and child were attacked after eating the pie, was affected, notwithstanding that she took none of it herself. It was, however, found that the knife which was used to cut up the pie was also used in helping her to dinner.

From investigations of the different cases of food poisoning that have occurred, the following deductions may be drawn :—

(*a*) In food which has become poisonous by keeping, one or both of these two conditions obtain—viz. a living microscopic organism, and an organic chemical poison, which may be a ptomaine, albumose, or toxin.

(*b*) The substance which is the immediate cause of the production of the morbid phenomena or symptoms is the chemical poison, which probably is produced by the action of the micro-organism on the albuminous constituents of the food. In the majority of cases the presence of oxygen appears to be necessary for the development of poisonous ptomaines, and, therefore, finely-divided meat is more liable to become toxic on account of the large surface which is thus brought in contact with the oxygen of the air. In certain of the German sausages, liver and blood enter into their composition, and these ingredients appear to be especially prone to develop poisonous bases.

(*c*) Provided the conditions are favourable to the growth and activity of the micro-organism contained in the article of food, it may produce its own peculiar chemical poison from the material affording it nourishment, either outside the body of man or within it.

(*d*) Both the specific micro-organism in an infected food, and the poison resulting from the fermentative and putre-

factive changes induced by it, may be fleeting as regards their existences, since the micro-organism may be killed by its own products, or the chemical poison, from its unstable nature, may undergo decomposition; so that an infected food which may be poisonous at one time may fail to be poisonous at a later period.

(*e*) In many of the cases of food poisoning that have occurred, an incubation period has been noticed, although in some cases it is practically wanting. When an incubation period occurs, in all probability that period is being employed by the micro-organism growing and cultivating itself within the human body, and producing its poisonous chemical substance, to which the symptoms arising at the end of this period are no doubt due.

When symptoms are produced by poisonous food without an incubation period (that is, in from half an hour to a few hours after taking the food), they are probably due to the action of an organic chemical poison previously manufactured in the food. This is what one would naturally infer, since a micro-organism, as in the other specific infections, would require time to grow, and to develop its chemical poison; whereas an article of food containing an already developed chemical poison would naturally operate more speedily, and the rapidity of the action of the poison would depend upon the amount of it taken, its poisonous nature, and on the idiosyncrasy of the individual taking the poisonous food.

(*f*) That the symptoms induced by poisonous food are not always due to chemical poisons previously produced in the article consumed, but that they are due to a true infection, is shown by the fact that extremely virulent micro-organisms have been found in articles of food, and in the viscera of persons dying from the consumption of such articles. As was shown in the Portsmouth pie-poisoning case, some of these bacilli may not be pathogenic or disease-producing on inoculation, though when taken by the mouth they may produce a chemical poison, which, absorbed from the alimentary canal, is capable of producing

illness, and even death. The not uncommon practice of eating high game is one which is liable to be attended with danger, since the highness of the game is dependent upon putrefactive changes that have occurred in it. The cooking of the game possibly destroys the bacilli of putrefaction in the food, but does not necessarily destroy the chemical products of the putrefaction; hence, after consumption of such game, a certain amount of headache, nausea, lassitude, and frequently slight diarrhœa, are not uncommon symptoms the next day.

As to the nature of the chemical poisons produced in articles of food by bacterial agencies, the following three classes are known:—

1. The ptomaines or animal alkaloids resulting from putrefactive or fermentative changes.

2. The albumoses or poisonous proteids, which are also produced by bacterial agencies, and probably occupy an intermediate position between albumins or proteids and ptomaines.

3. Toxins or poisons possessing an uncertain composition, and which are also probably produced by bacterial agency.

II. *Fish.*—Cases of poisoning by fish, and especially by certain kinds of shell-fish, are not uncommon. Mussels, crabs, lobsters, oysters, salmon, and sardines may produce symptoms of poisoning. The ordinary symptoms are those of irritation of the stomach and intestines, with vomiting and diarrhœa; more or less nervous disturbance occurs, not uncommonly accompanied by a nettle-rash. Fatal effects may result owing to fish becoming toxic from the development of a chemical poison by bacterial agency, and in some instances the bacilli or germs appear to grow practically without an air-supply, as most probably occurred in connection with a fatal case of poisoning by tinned salmon that was investigated by the author in 1891, and a fatal case of poisoning by tinned sardines that was investigated by Stevenson in 1892. In the former of these cases the fish, to appearance, taste, and smell, seemed to be perfectly sound, and was consumed without suspicion. In the latter case, reported by

Stevenson,[1] a military officer, aged twenty-one years, took six hot sardines on toast for breakfast; during the day he vomited twice, and the following morning was seen by a medical man, when he was found to be suffering from slight pain in the stomach, a weak pulse, and stiffness and feeling of weight in the right leg; about an hour later he died somewhat suddenly, and it was then noticed that the right thigh and scrotum were œdematous. At the post-mortem examination made the next day, putrefaction was considerably advanced; the mucous membrane of the stomach and the rugæ of the intestines were emphysematous. The liver was friable and hyperæmic, as were also the kidneys and bladder; the endocardium was blood-stained. Stevenson found that the sardines left in the tin had no unusual appearance; their odour was peculiar, but not offensive; the tin itself was bright and uncorroded. Some of the fish were intensely toxic, killing mice and a rat fed on them. From four of the fish, weighing two and a quarter ounces, Stevenson extracted $\frac{1}{65}$ of a grain (1 milligramme) of an alkaloidal substance, two-thirds of which, injected beneath the skin of a young rat, caused its death within four hours and a quarter. No characteristic pathogenic forms of bacteria were discovered in the sardines. A piece of one of the sardines, which, when eaten, had caused the death of two mice, was macerated, whilst swarming with ordinary bacteria, in sterilised water, and placed beneath incisions in the backs of two guinea-pigs, both of which were unaffected. These experiments pointed to a toxic ptomaine, and not to the direct action of pathogenic bacteria on the animal organism. From the contents of the stomach, and from the vomit of the deceased man, small portions of an alkaloidal substance were extracted which, injected into the backs of young rats, killed them. Stevenson was of opinion, from the results of his experiments, that the case was one of ptomaine poisoning, and he inclined to the opinion that the ptomaine was generated before the tinning of the sardines.

Most probably in these cases the contents of the tins were

[1] *Brit. Med. Jour.*, 1892.

not completely sterilised, and so, after sealing, the micro-organisms continued to grow and produce a chemical poison without a supply of air.

Many cases of poisoning from eating mussels have occurred. Formerly it was believed that mussels owed their poisonous properties to the presence in them of copper derived from ships' bottoms; but the researches of Brieger have shown that the poisonous effects are due to a ptomaine named mytilotoxine, a poison which is produced within the mussels by bacterial agency, the bacteria generally gaining access to the fish from sewage contamination of the water in which they are living. It has been found that mussels gathered in harbours or on shores polluted by sewage are not unfrequently poisonous, and contain the ptomaine mytilotoxine; whereas, if some of these mussels are taken to the open sea and laid down there for a few months, they lose their poisonous properties, and cease to contain mytilotoxine. If the mussels inhabit stagnant water, or water containing decomposing matter, they may also develop their peculiar poison. Oysters also, if grown in water to which sewage gains access, may develop poisonous properties.

Cameron[1] relates the cases of seven persons who, after partaking of a meal of stewed mussels, were seized with severe symptoms of poisoning; five of the affected persons died. About twenty minutes after the ingestion of the mussels some of the children affected complained of a sensation of 'pins and needles' in the hands; these sensations were rapidly followed by vomiting, dyspnœa, swelling of the face, want of co-ordination in movement, and spasms, principally in the arms. One of the children died in less that an hour, the mother and three other children succumbing within two hours after eating the mussels. The patients apparently died of asphyxia, their faces being intensely livid. The mussels had been procured from a small sheet of water to which the sea had access, but which received fresh water and some sewage. McWeeney obtained, from

[1] *The Lancet*, 1890

mussels taken from the same pond, cultivations containing numerous colonies of bacilli.

Todd[1] records a case of poisoning by mussels which occurred in an elderly man, and which, under treatment, was followed by recovery. The patient partook freely of mussels one evening, and about two hours later he felt giddiness coming on, followed by a prickling sensation in the hands, extreme dryness of the throat, colicky pains, and diarrhœa; loss of sight and unconsciousness quickly followed. When seen shortly afterwards he was in a state of collapse, with small quick pulse, and lividity of the face, particularly marked about the nose; he was conscious, but there was loss of speech and sight, and profuse diarrhœa. When able to speak he complained of acute pain in the epigastrium and at the nape of the neck. The treatment consisted in producing vomiting by an emetic of mustard, applying warmth externally and mustard poultices to the epigastrium and nape of the neck, and the subsequent administration of stimulants, including digitalis, together with ten drops of the *liquor strychninæ* every half hour till relieved. In four hours he had recovered from the graver symptoms. Cameron[2] records some cases of apparent poisoning by oysters, in which the symptoms were nausea, vomiting, diarrhœa, abdominal pain, and severe prostration; no deaths resulted. The oysters seemed to be quite fresh.

In tropical regions certain of the fish are exceedingly poisonous when used as food, either at certain seasons, or perhaps in the case of some species always. Several deaths occurred on a steamer[3] during a voyage from Rangoon to Marseilles, from some of the crew partaking of some fish which had been caught in tropical waters; several of them died in terrible agony.

III. *Milk.*—Milk may produce symptoms of poisoning or of disease in several ways. It may become acid from lactic acid

[1] *Brit. Med. Jour.*, 1891. [2] *Ibid.*, 1890.
[3] *The Lancet*, 1892.

fermentation, and in this condition is extremely liable to produce flatulence, sickness, and diarrhœa in children. Milk may also develop a poisonous substance named tyrotoxicon, which was first discovered by Vaughan in poisonous cheese. In addition, milk may contain the germs of several of the specific fevers (see p. 414).

IV. *Cheese.*—Under certain conditions cheese may become poisonous, due to the production, as the result of a peculiar fermentation, of a toxin discovered by Vaughan and named tyrotoxicon. Tyrotoxicon was at first considered to be a ptomaine, but its chemical composition is that of diazo-benzene-butyrate. This substance is an extremely powerful poison, producing nausea, dryness of the mouth and throat, vomiting, diarrhœa, and extreme nervous prostration. Usually the symptoms pass off after a few hours, but death may ensue from collapse. Milk and cream, as well as cheese, may contain tyrotoxicon. Cheese containing this poison need not be, either in appearance or taste, different from that of sound cheese.

V. *Vegetables.*—Vegetables may also develop poisons as the result of putrefactive changes, although this accident is not so likely to occur as is the case with the different kinds of meat and fish.

2. **The poisonous results may be due to the presence of toxic metallic salts.**—Tinned provisions, but especially tinned fruits containing an acid juice, may produce poisonous symptoms from impregnation with soluble tin salts. These are probably produced by the malic acid present in the juice exerting a galvanic action upon the solder of the tin, and so carrying some of it into solution as malate of tin. Tinned cherries, apples, pineapples, and tomatoes are the most liable of the tinned provisions to contain tin, the acid juice acting upon the solder of the tin as just described, or possibly on the tin surface, owing to the interior being coated with impure tin. In 1890 four cases of tin poisoning, caused by the consumption of tinned cherries, were investigated by the author, and, owing to the tin from which the patients had eaten being left half full, a determi-

nation of the amount of soluble tin salts present, and therefore of the amount that had been taken in each case, was effected. The juice contained in solution malate of tin equivalent to nearly two grains of the higher oxide of tin in each fluid ounce. It was estimated that the symptoms (in each case occurring in robust men) were produced by doses of malate of tin varying from four to ten grains. Two of the patients nearly died from the severity of the diarrhœa and collapse, but, finally, under treatment, they all recovered.

Copper sulphate is occasionally used to give a rich green colour to preserved peas and other vegetables, to olives, and to green pickles; copper may also gain access to articles of food or to preserves which have been kept in the cold in copper vessels exposed to the air. The colouring of vegetables by the use of a copper salt is due to the formation of a copper compound with the phyllocyanic acid derived from the chlorophyll. This copper salt, being an insoluble one, is not as a rule found in the liquid surrounding the peas, &c., but all the same, when such peas, &c. are consumed, the copper compound can exercise a detrimental effect.

Water may become contaminated with lead derived from leaden pipes, or from lead-lined storage cisterns (see p. 199). Copper and zinc may also occur as impurities in drinking-water.

3. **The poisonous results may be due to the presence in the food of the germs or spores of certain specific diseases.**—In this way such specific diseases as typhoid fever, cholera, scarlet fever, diphtheria, tuberculosis, actinomycosis, and anthrax may be conveyed to man. Water is the usual medium by which the typhoid germ is conveyed to the system of man, owing to the pollution of water with sewage containing typhoid stools. The dissemination of cholera is also mainly due to the contamination of water with cholera bacilli derived from cholera stools. Milk may contain the bacilli of typhoid fever, cholera, scarlet fever, and diphtheria. The typhoid germs may gain access to the milk by polluted water either being added to the milk, or by its being used to cleanse the milk cans, or by ex-

posure of the milk to sewer air. The germs of scarlet fever may be conveyed into the milk either from the hands of a milker suffering from scarlet fever in the peeling stage, or from cows suffering from a disease which is either identical with, or closely resembles, human scarlet fever. Diphtheria is possibly conveyed from the cows themselves. Tubercular disease of the intestines, which is so common among young children, is probably due to the milk containing tubercle bacilli derived from tubercular udders of cows. If such milk be given unboiled to children, these tubercle bacilli are capable of setting up tubercular ulceration of the intestines. Milk may also be contaminated through the animal suffering from foot and mouth disease; if vesicles are present on the teats of cows suffering from this disease, the virus may gain access to the milk, and then is capable of producing a somewhat similar disease in the person consuming such milk. The rare disease known as actinomycosis is probably due to eating raw barley or other cereals upon which the actinomyces fungus has grown.

4. **The food may be infected with certain parasites or their ova, which parasites, or ova, are capable of undergoing development in man.**—Pork may contain the small trichina spiralis worm, and so may produce in man the disease called trichinosis. The trichina of pork is a small worm about $\frac{1}{30}$ of an inch long, coiled up in a fibrous capsule between the muscle fibres; when swallowed in this encysted form, if the trichinæ have not been killed by proper cooking, the capsule dissolves in the digestive juices, and the worms are set free and mature and pair in the intestines in about three days. The ova are hatched within the uterus of the female worm, and the birth of the embryos begins on the seventh day, and continues for some time; they migrate through the intestinal walls mainly by direct passage, and also by gaining access to the lymph and blood streams, and finally settle down in the muscles, where they become encysted.

Pork may also contain the cystic stage of the tape-worm and so give rise to the tape-worm, tænia solium, in man; such pork is commonly known as 'measly' pork. Beef may contain the

cystic stage of the tape-worm, tænia mediocanellata, and so give rise to that tape-worm in man.

Vegetables which are eaten by man uncooked, such as lettuces, watercress, &c., may contain on the surface the ova of a small tape-worm, the tænia echinococcus, which inhabits the intestines of dogs; the presence of the ova on the vegetables is due to dogs defæcating thereon. Such ova introduced into the stomach of man are the cause of hydatid disease. Water may also contain these ova from contamination with dogs' fæces, and may so give rise to hydatid disease in man.

In connection with several of the cases of food poisoning described, it will have been noticed that the food frequently appeared sound and good, so far as could be judged by appearance, smell, and taste. In such cases it would be obviously unjust to hold the manufacturer or vendor of such articles of food responsible for any disastrous effects following their consumption, unless it can be shown that unsound meat, &c. was used at the outset, or that there was gross and culpable neglect of the ordinary precautions as to cleanliness at the place or places where the food was prepared. The exact degree of responsibility attaching to the manufacturer or vendor is a matter that must be decided with special regard to the circumstances of each particular case.

In 1893 several cases of brawn-poisoning, with two deaths, occurred at Burry Port; the coroner's jury committed the butcher, who supplied the meat, for trial on the charge of manslaughter; at the conclusion of the trial he was acquitted.

END OF VOLUME I.

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